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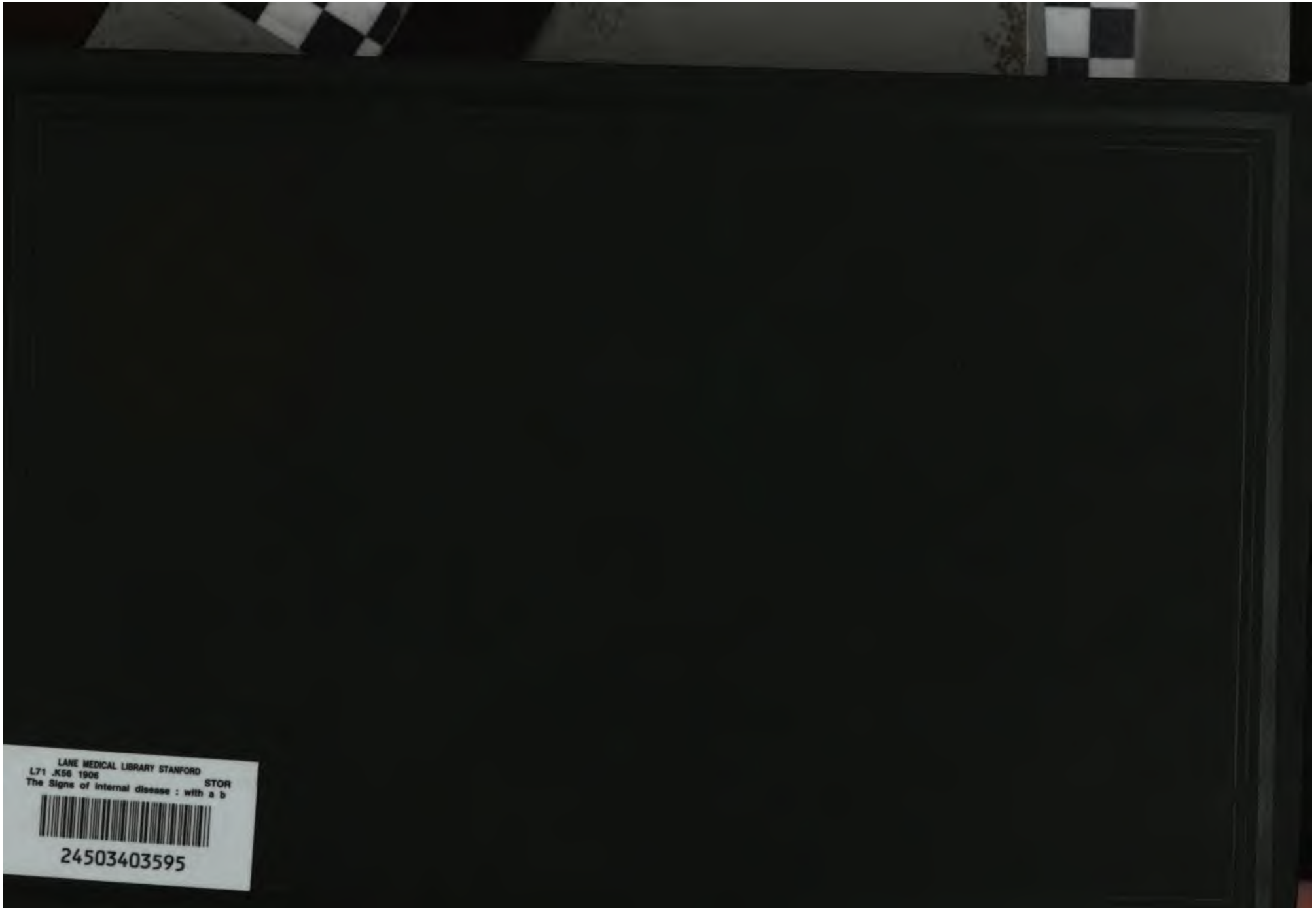
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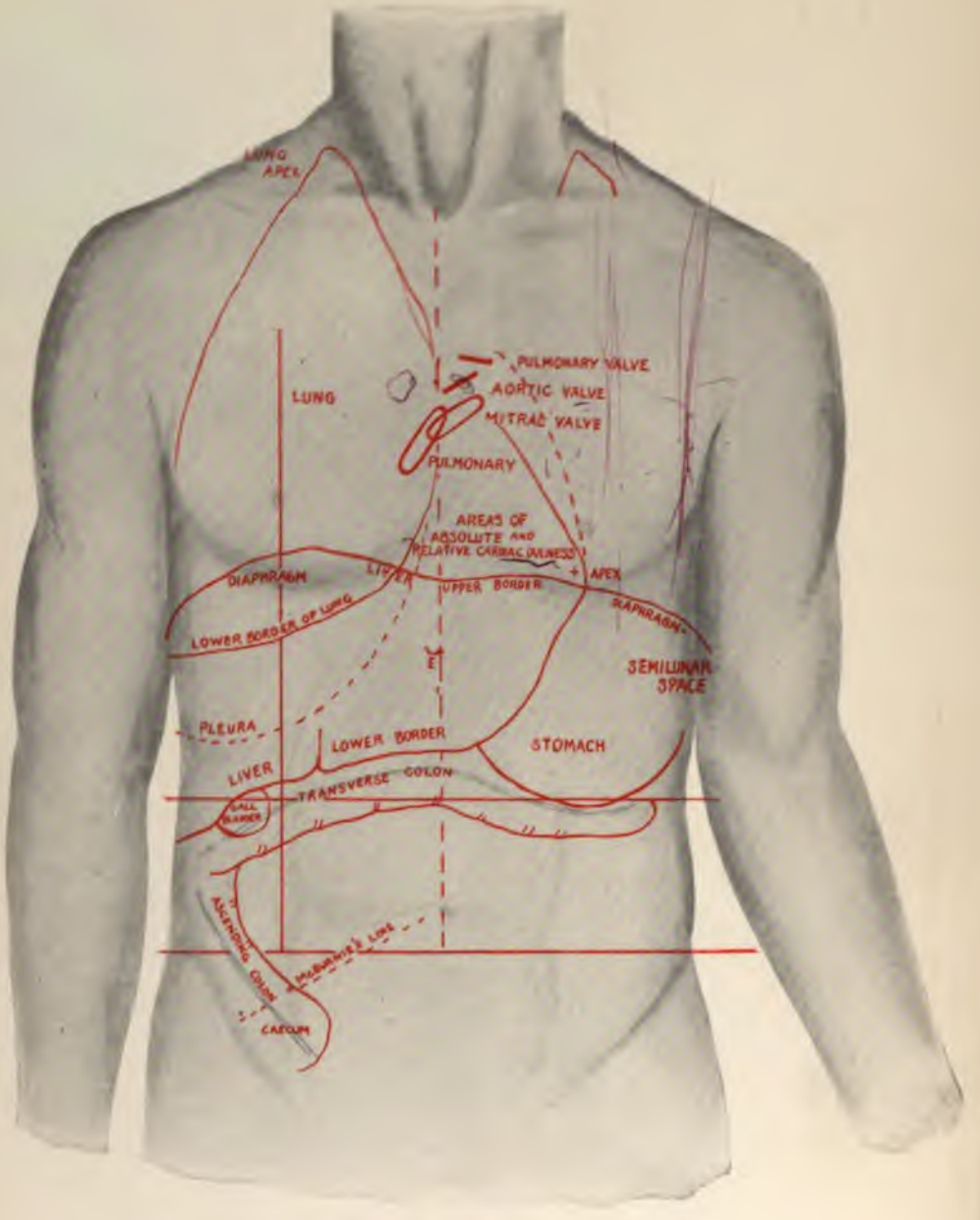

FRONTISPIECE.

Surface Topography During Moderate Expiration.

THE SIGNS OF INTERNAL DISEASE

With a Brief Consideration of the Principal Symptoms thereof

BY

PEARCE KINTZING, B. Sc., M. D.

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ILLUSTRATED

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To

Richard Armstrong, A. M., M. D.

In remembrance of kindly help and sympathy
during my earliest efforts to acquire
the science of medicine,
and as a tribute to his fifty-four fruitful years of practice,
this book is respectfully dedicated.

54329

PREFACE.

This volume is the outgrowth of my lecture notes. First intended for the students of my classes at the college and hospital, it has grown gradually to its present proportions, when it seemed worthy of wider circulation.

While little that is new or original can be said upon certain of the subjects which have been so well worked over by Da Costa, Loomis and Gairdner, who stand in their relation to physical diagnosis in the position occupied by the old masters in the field of art, yet the value of putting one's ideas and knowledge of a subject into concrete form for teaching so far surpasses the endeavor to teach from the abstract, that I offer no other excuse for being chargeable with the offense implied in Cadmon's—"Of the making of books there is no end."

Most books upon technical subjects are written by the men of the schools and the laboratories, who, with the endless resources at their command, are apt to give small consideration and less preference to methods better suited to the class of the general profession which might almost be called its lay-members.

I have strongly endeavored to keep these in mind and to choose always the simpler way when it sufficed.

I am much indebted to Miss Margery Comegys for artistic work and help on the plates and illustrations and to Dr. F. W. Hachtel for assistance in proofreading and indexing.

PEARCE KINTZING.

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SECTION I.

DISEASES OF THE CHEST.

INTRODUCTION.

It is in diseases of the chest that physical research has reached its highest development and has been most prolific of results. It may be claimed that the science of physical diagnosis cannot be learned from books. For reply, we ask,—what science can? Nevertheless, the orderly setting forth of the groundwork, and a clear description of the phenomena on which are based the inferences and conclusions of physical diagnosis are as necessary and helpful to the students as is the same work in any other department of science.

Yet, withal, that the ability to make correct application of these principles and more especially the acquisition of capacity to interpret their import and significance, must be gained by bedside practice, is freely admitted.

The manner of investigating disease of the chest should be taken by the student as a type of the methods used to investigate disease in other parts of the body. It is absolutely necessary for the student first to make himself thoroughly familiar with the normal sounds and normal properties connected with the functions of the thoracic organs, and to fix firmly in mind their exact location and natural anatomical outlines. Experience has taught me that these deficiencies are the great stumbling blocks of the student. This knowledge can be acquired by painstaking study upon the living subject, combined with the practise of the same methods upon the healthy individual that would be used were he diseased, until these normal properties are a part of our consciousness, and departures therefrom as quickly recognized as is a false note by the *maestro*.

It must be remembered that departures from normal sounds and changes in normal areas are caused by alterations in the physical properties of the organs or parts affected, and in no other way. As often as we strike the key of an organ we elicit a certain invariable note, and it is only by altering the physical conditions or the mechan-

ism at some point between key and pipe that we can change the character of the sound. So the principal thoracic organs, in the accomplishment of their functions, produce certain fixed sounds which are altered only by changes in their physical properties or in the mechanism of the sound production. To determine the extent and significance of these alterations is a part of the office of the diagnostician.

Further than this, all matter is endowed with certain individual properties, and to make manifest the different degrees in which various organs may possess one or another of these, is also a part of physical diagnosis. Thus, the capability of producing resonance, of transmitting or conducting sound, of vibrating when struck, varies materially with the different tissues of the body, and all of these properties are profoundly affected by disease, by alterations in their structure, or by changes in their surroundings. Yet disease must, indeed, be extensive ere a structure loses entirely its normal properties. It is manifest that as long as any of the normal structure remains unaltered, that far will it retain its normal properties. On the other hand, these will be appreciably or materially altered in some cases by even slight disease; hence diagnosis becomes, broadly speaking, a question of estimating differences or departures from the normal. Just as the ear of the better trained musician is able to recognize even the finest shading of a note, so the person more skilled in recognizing minute differences in the physical signs which it is possible to elicit from the human body will be, other things being equal, the better diagnostician. But the work does not stop here. It is the ability to correlate, to judge the relative importance of the facts brought to light, and to draw rational inferences therefrom, which marks the true diagnostician.

These refinements can be learned only by practice and perseverance. No description suffices. Only by comparisons, by comprehensive knowledge drawn from wide sources, can anything resembling perfection be reached.

Gairdner truly says, "The popular, and to a certain extent the half-educated, medical mind is always looking for a pathognomonic sign, or a broad, striking, easy generalization from a few facts; whereas it is only by ripened experience that we come to know gradually the real value of common and obvious, still more of uncommon and not obvious, facts *when seen in combination*, so as to form conjointly a basis for large inferences."

METHODS.

To the end that we may attain the objective point as above set down, it is of the highest importance that we proceed along orderly lines. The various steps of the examintion bear a certain relation to each other, as to sequence, and this law of harmony should not be violated heedlessly. Again, I emphasize the dictum that it is only by minute, painstaking repetitions, by careful comparisons of suspected with healthy structures, that we may hope to arrive at reasonable certainty. A hasty conclusion, jumped at in one step of the examination, unconsciously may pervert the judgment as to other palpable signs, that these may bear out the false conclusion. It is generally agreed among workers in this field that the most orderly progression is the one here to be followed.

First, a clear, succinct *history of the case* should be obtained, keeping before the mind the leading factors of what constitutes useful and necessary information, never approaching the case with a preconceived idea of what *ought* to be found. Nothing, perhaps, better illustrates the comparative tact and talent of different examiners than their ability or inability to obtain leading facts. Much care is necessary not to suggest the answer. It is well established that the complaisance of a large class of patients prompts them to give the answer which they believe is expected. This is especially true of hospital and dispensary habitués. Too much cannot be said of the present and future value of correct case records. That doctor's life must indeed be busy, who has not the time to devote to this work. In examining cases, a physician is necessarily guided by the circumstances in which he finds the patient, as well as by his knowledge and experience of the condition with which he is called upon to deal; hence, no single method is applicable to all cases. We begin our inquiry by ascertaining what the patient feels to be wrong. This usually refers us to some one organ or part, and the local investigation is followed by a general survey of allied functions or organs, with inquiries into the manner in which the present illness began, the previous health and kindred questions, the family history, the previous history and the present state of the patient.

It is well to separate the manifestations of the disorder into two divisions: subjective, or those of which the patient is conscious, and objective, those of which the examiner gains knowledge by his own senses. The former are classed as *symptoms*, and the latter as *signs*

or physical signs, a useful but often disregarded classification. Further, it is simple and natural to combine to a certain extent anatomic and physiologic facts in related organs. Thus the respiratory and circulatory systems are not only closely allied, but have their seat in the thorax; while the genito-urinary system and the various organs of digestion are situated in the abdominal cavity, and may be conveniently grouped consecutively.

We begin with that system which, from the general aspect of the case, seems to be the chief defaulter, and give it the fullest consideration. Thus, as suggested by Sir William Roberts, "If we find orthopnœa, general anasarca, and distended jugular veins, we begin with the circulatory system. If we find purulent expectoration, emaciation, and clubbed finger-ends, we begin with the respiratory system. If we find albuminous urine and pale, puffy countenance, we begin with the urinary system," etc.

Few writers on medical diagnosis, and fewer still of the systematic writers upon medical practice, offer the student any substantial aid upon this important subject of case investigation. Apart from hospital internes, the great body of students is left to work out its own salvation, and devise a method as the result of experience. Experience means waste. Hence we append a short synopsis, modified to suit our present purposes, derived chiefly from the plans proposed by Sir William Roberts in his work on *Practice*, and the form proposed by Leach, as elaborated by Finlayson, from which source also some of the preceding suggestions were obtained.

Name. Sex. Age. Address. Occupation. Date. Preliminary inspection; general information; definite inquiry as to what has brought the patient to seek advice; (incorporating the patient's own statements in so far as they are relevant.)

Previous History.	*Present Condition.*	*Diagnosis.*
1. Social.	1. External.	
2. Previous Health.	2. Special Organs.	
3. Present Illness.	3. Circulatory System.	
4. Family History.	4. Respiratory System.	
	5. Digestive System.	
	6. Genito-Urinary System.	

Under the first division we place the leading facts in order named:

1. *Social.* Residence—*climate*—prevalence of special diseases—*occupation*—exposure (heat, cold, chemical products, dust,)—food—stimulants—tobacco—drugs. Mode of life—facts about marriage—children—parents.

2. *Previous Health.* Nature and character of previous illnesses, (confined to bed—hospital—interruption of occupation)—Indications pointing to special affections—especially rheumatism—syphilis (adult and infantile)—cough—hæmoptysis—anæmia (probable cause)—sexual disorders—gain or loss of weight.

3. *History of Present Attack.* Onset—cause of symptoms—treatment if any.

4. *Family History.* Age and causes of death—special diseases which have shown themselves.

Under the second division, PRESENT CONDITION, are noted most of the signs observed or ascertained by the examiner, which we have classified as objective. Of necessity some of these signs fall into both categories, but are classed here owing to their greater significance. These points, in so far as they have special reference to our own subject are fully dwelt upon under the sections on Inspection, Ausculation, etc.

1. *External Surface.* Posture,—temperature—appearance—color of skin—expression (languid, sallow, worn, wasted, flush, anæmic)—nostrils—lips—arcus senilis—conjunctivæ—state of nutrition—senility—presenilty. Peculiarities of development—deformities—joints—tumors—swellings. Skin—œdema (where)—perspiration—rough—dry—cicatrices—rashes—discolorations—superficial vessels (especially of neck, abdomen and extremities). Hair—nails—finger-tips. Glandular enlargements (where).

2. *Special Organs.* Disorders of vision—of hearing—(ear discharges)—other senses—with special details as they bear upon the case.

3. *Circulatory System.* Palpation—Cardiac pain—dyspnœa—pulse—location of apex impact—area of impact—character—local bulging—neck—pulsation. Other regions. Venous engorgement—pulsations—cyanosis—thrill (where)—area of percussion—dullness. Results of auscultation at four cardinal points. Murmurs. Blood examination.

4. *Respiratory System.* Number and character of respirations —dyspnœa (inspiratory or expiratory)—orthopnœa—effect of exertion—pain—cough—sputa—voice—larynx—throat. Results of inspection, palpation, percussion, auscultation and mensuration, with due attention to details mentioned hereafter in the special sections, carefully describing and exactly locating all alterations—the conduction of heart sounds—the resistance. New sounds.

5. *Digestive System.* Lips—teeth—gums (red or blue lines)—tongue. Fauces. Thirst—appetite—pain or discomfort after eating —flatulency—acidity—hiccough—vomiting (character).

Abdomen—walls—pain—tenderness—colic—ascites—state of bowels—character of motions—abnormal constituents (worms, blood, pus,)—piles.

Liver—size—feel. Spleen—size—feel. Hernia—other regions.

6. *Genito-Urinary System.* Frequency of micturition—pain—difficulty. Special examination of urine—quantity in 24 hours—color —reaction—specific gravity—clearness—albumen—sugar—urea—deposits. Microscopic examination and results.

Males, strictures—specific diseases.

Females, menstruation—its disorders—leucorrhœa—pregnancy—specific diseases.

For detailed methods pursued in the examination of the nervous system the reader is referred to special works on that subject, in which they are usually fully exploited. Such notes as bear upon the special case may be inserted conveniently at the end of the above scheme.

The order in which the steps of the physical examination should follow each other has already been intimated in the above plan. Definitely stated they are:

(a) Inspection;
(b) Palpation;
(c) Mensuration;
(d) Percussion; and
(e) Auscultation.

These complete the case record and bring us face-to-face with the momentous conclusion for which our work has been undertaken,—the *Diagnosis.*

In all cases in which a justifiable diagnosis can be reached it should be recorded, as it engenders greater care on the part of the

examiner and carries a certain weight of responsibility. Such a record does not preclude a subsequent revision of opinion, and certainly it more definitely shapes the last step,—the *Treatment*.

The first requisite of diagnostic science is a good *stethoscope*. While almost any instrument can be made to answer, yet comfort in

Fig. 1—Camman's Stethoscope.

the use of the instrument, as well as satisfaction and refinements in the results of its use, vary widely with the choice. Avoid cheap, badly-made instruments, as well as heavy, cumbersome ones. See that the ear tubes fit naturally and easily into the ears, and that they perfectly close the canals. A large ear-bulb is more satisfactory than a small one, especially for protracted use.- A metal spring is more satisfac-

tory than gum elastic, for retaining the instrument in position. A too weak spring is annoying; a too strong one fatiguing. The conducting pipes *should be of the same caliber throughout,* and not too small. The abrupt shoulder of the jointed stethoscope is an objection. The bell should not be too large, since a large bell does not admit of being evenly placed against the tissues, especially in the intercostal spaces, and thus extraneous sounds are admitted, which, being conducted to

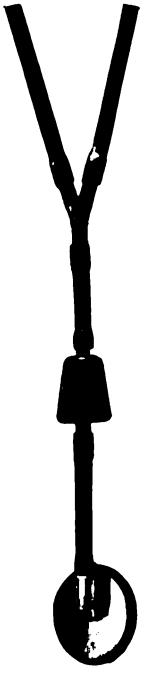


Fig. 2—Bowle's Stethoscope, Compound Form.

the ear of the auscultator, cause confusion. The soft rubber supplementary bell, devised to correct this defect, is not wholly a success.

The binaural stethoscope has replaced entirely the single instrument. That devised by Dr. H. K. Valentine, of Brooklyn,* is one of the best types with which I am familiar, and possesses the requisites named. The Bowles instrument is very satisfactory. The ear pieces do not differ from those in ordinary use: two pieces of rubber tubing

*Described in the *New York Medical Record* of July 16, 1892.

attach them to a hard rubber Y, which is again joined by rubber tubing to the drum. The drum is a steel disc somewhat larger than a silver dollar and about twice as thick. It is pierced in the center by a small hole into which fits the elbow-joint pipe which connects it to the Y-piece. The face of the drum is slightly cupped, and over this concave face fits a thin, hard rubber disc which receives the sound

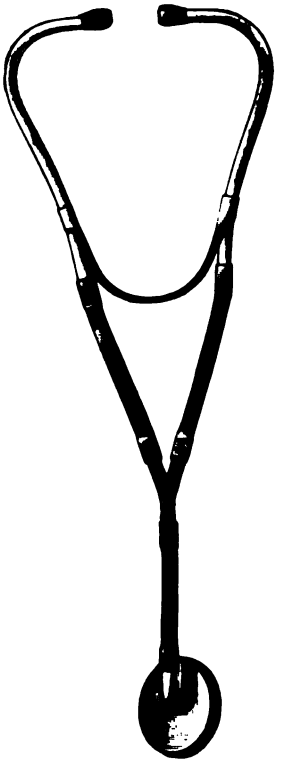


Fig. 3—The Bowle's Stethoscope.

vibrations. The sounds are conducted with extraordinary clearness and intensity, and its use is especially commended to those whose sense of hearing is less acute than normal. The flat disc admits of its being slipped under the back without disturbing the position of the patient, and thus may be employed when his condition forbids the use of the common type, as during or immediately after severe hæmoptysis, in which a recent experience of my own was instructive. With its use,

breath sounds may be heard in areas where otherwise they are inaudible, as in the subscapular region. The drum may be removed and the ordinary bell be substituted when desired.

Fig. 4—Phonendoscope.

The phonendoscope, of Bazzi and Bianchi, is cumbersome, expensive and possesses few, if any, advantages over the one described. It is excellent for auscultatory percussion, whereby the outlines of the various organs may be mapped out with great certainty.

SECTION II.

MEDICAL ANATOMY.

SHAPES AND TYPES OF THE CHEST.

The shape of the thorax and its general capacity as well as its expansile powers, are influenced by age, sex, occupation, heredity and disease. Heredity impresses its most striking as well as its strongest peculiarities upon those structures which earliest develop and earliest reach maturity, hence the bony skeleton is strongly influenced thereby.

At birth the contour of the chest is almost circular and the thorax cylindrical, but as development proceeds the expansion and growth of

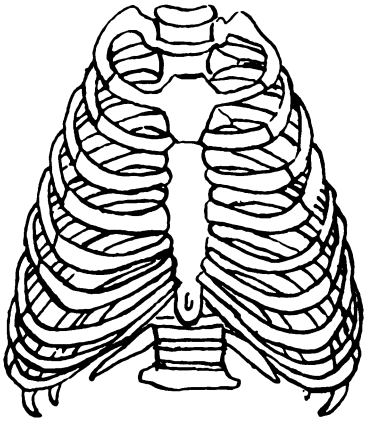


Fig. 5—Thorax of Infant, Showing Conical Shape.

the lungs push out the ribs in the lower lateral areas, the chest flattens and at the same time becomes conical. As depicted by Morris, the axillary border of the chest at the eighth month shows little or no vertical curvature. The relative shapes of mesial transverse vertical sections of the infant and adult chest are illustrated by the accompanying figure.

In old age the shape of the chest by slow retrogression approaches

once more the cylindrical type. Here again the most marked changes take place in the inferior areas; the lower circumference showing considerable recedence while the middle zone changes least. Atrophy of the muscles of the shoulder girdle occurs with age, and the resulting

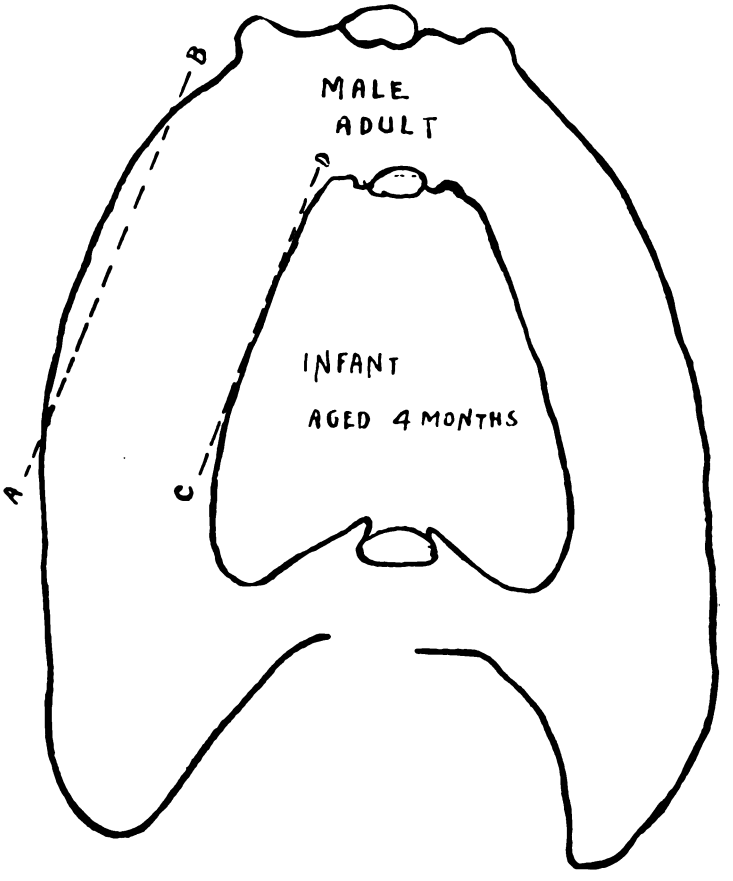


Fig. 6—Vertical Mesial Sections Adult Chest and Infantile Chest. The lines AB and CD are parallel.

stooping posture gives rise to a seeming contraction of the upper chest zone which in reality is very slight.

Types of Thorax.—Defective development combined with inherited tendencies aided by malnutrition and imperfect expansion produce the most common of the vicious types of chest. Such are the

rickety chest and the variations thereof known as the "pigeon-breast" and the phthisical or paralytic chest. In its most modified degree the rickety deformity consists simply of a flattening of the lateral areas of the chest with corresponding decrease in the transverse diameter. This results in a degree of forward bulging of the chondral area, which is spoken of as transverse constriction. This and the succeeding forms originate when the bones are still soft and pliable, and in general are associated with deficient expansion, which is not only a cause but a permanent result of the condition.

In true rickets the chest is flattened laterally and its contour is trapezoidal, with the wide border posteriorly. Just outside the junction of the ribs with the cartilages there is an oblique shallow depression extending downwards and outwards, which transverse constriction makes the sternal portion of the chest unduly prominent. A shallow groove or curve passing outward from the level of the ensiform cartilage towards the axilla is known as Harrison's sulcus, or curve. It corresponds with the attachment of the diaphragm to the cartilages and is more prominent and deeper during inspiration. Posteriorly the spine shows vertical curvature. The line of junction of the ribs with the costal cartilages presents small, nodular enlargements, generally visible, always palpable, which appear as early as the third month and disappear about the fifth year. They are fantastically called the "rickety rosary." The bones are spongy and increased in size. Expansion is impaired.

This deformity is not peculiar to rickets. Other conditions which interfere with free respiration produce the same deformity, particularly enlarged tonsils, as pointed out by Dupuytren in 1828. This type of chest is particularly interesting owing to the predilection which its possessor has for respiratory disease.

Laryngismus stridulus and other spasmodic affections of the larynx are also of frequent occurrence.

The *pigeon or chicken breast* is an exaggerated type of the above. The antero-lateral constriction reaches such a degree that the sternum, especially the lower half, is pushed far forward and a transverse section of the thorax appears nearly triangular. The curvature of the ribs lies almost entirely at their angle, whence they project nearly straight forward to the sternum. The lower ribs are flared out by the liver and Harrison's sulcus is well marked. The lower and lateral regions of the chest retract with inspiration. The causes which pro-

duce the rickety chest are responsible for the chicken breast and the tendencies are the same.

The Funnel Breast. This name has been bestowed by the Germans("trichter brust") upon cases which present marked recession of the ensiform cartilage. The name does not apply to cases in which the cartilage has been bent inward the result of occupation, but to those in which respiratory obstruction has resulted in its permanent

Fig. 7—Types and shapes of chest. Acquired (occupational) deformity. The interspaces on left side are much wider than on the right.

retraction by the act of breathing. In such cases the lower sternal region and intercostal angle is distinctly excavated and retracts during *inspiration.* Enlarged tonsils, whooping cough and many respiratory affections are assigned as causes.

The Barrel Chest. This deformity is also called the emphysematous chest. It may occur as early as the tenth year, but is generally seen only in adult life. The chest becomes rounded and barrel-shaped, the intercostal spaces are widened, the neck is short and the back bowed, the shoulders are bent forward, the arms hang lax, giving a

stooping appearance to the possessor. The circumference as taken by the cyrtometer shows a close approach to a circle. In early life the change is often due to asthma, the result of naso-pharyngeal disease; in later life to emphysema, the causes of which are discussed thereunder.

The Phthisical Chest. While Hippocrates recognized a form of chest as predisposing to this ailment, yet a disease so universal is no

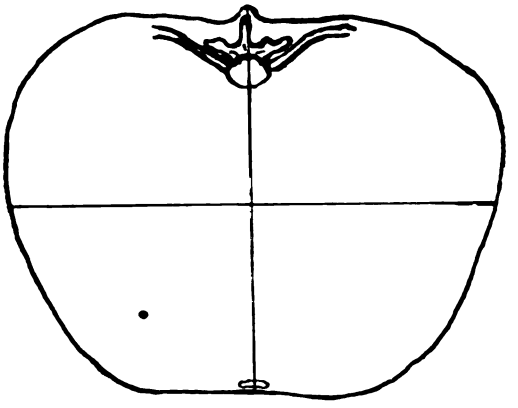


Fig. 8—A normal adult male chest outline. 6th costal cartilage.

respecter of types and it can scarce be said that the description holds good.

The form of chest usually described as phthisical is the *result* of the disease rather than one of its causes. Such a chest is long, narrow and flat with depressed sternum. The lung capacity is generally stated as being below normal, but Benneke asserts that this is a mistaken belief. The ribs slope downward abnormally, making an acute angle at the ensiform junction. All the fossæ are deep, especially the clavicular and suprasternal, and the scapulæ project wing-like from behind, hence the name—the "alar chest."

DIVISIONS OF THE CHEST AND ABDOMEN.

Diagnosticians divide the chest and abdomen into regions by imaginary lines and name the various sub-divisions for the most part in accordance with the underlying organs. Such sub-divisions are necessary for convenience in describing and locating the various organs as well as the lesions to which the parts are subject. Uniformity in this regard among systematic writers is most conspicuous by its

absence, these usually making their sub-divisions in accordance with fancied convenience.

The diaphragm separates the chest or thoracic cavity from the abdominal cavity. The diaphragm is attached anteriorly in the central line to the ensiform cartilage, to the lower border and inner surfaces of the cartilages of the six lower ribs. While posteriorly its crura are continued down over the bodies of the lumbar vertebræ the diaphragm as a partition extends only as far as the lower border of the ribs. It forms a vaulted roof to the abdominal cavity, arching on either side higher than in the middle portion where the pericardium is attached. It rises on the right side as high as the upper bor-

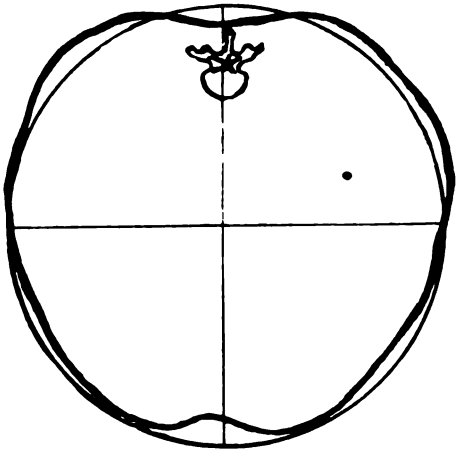


Fig. 9—Emphysematous chest of practically the same circumference as Fig. 8. Note approach to circle.

der of the fifth rib, and on the left as high as the top of the fifth interspace. Viewed from in front its line presents two domes, with a slight fall or "sag" in the middle.

MEDICAL ANATOMY.

Regions or Spaces of the Chest. While it is presumed that the student of physical diagnosis is thoroughly familiar with both the topographical and the relational anatomy of the parts studied, yet by reason of the aids to description which such anatomic depictions furnish to the writer, as well as for the ease of comparison and reference which they afford the reader, no work on the subject is complete which omits them.

The exterior of the trunk offers some well recognized landmarks which serve as cardinal points both for description and for location. Such are the clavicles, the nipples, the umbilicus and the ensiform cartilage in plain view anteriorly; and the median furrow or spinal gutter, the apex of the vertebra prominens, the spine of the scapulæ and the crests of the ilia behind.

The space above the clavicle is known as the *supraclavicular fossa.* The *infraclavicular space* lies below each clavicle, and while its upper border is sharply defined by that bone its lower border is separated from the adjacent mammary region only by an arbitrary line. This space extends inward to the sternum, outward to the shoulder, being bounded here by a vertical line bisecting the clavicle at its outer fourth. Emaciation throws the clavicles into strong relief and makes both the fossa and the space appear deeper and better marked. At the top of the sternum is the *suprasternal notch.* The *mammary region* extends outward from the edge of the sternum to the vertical line just located, and from the upper edge of the third to the upper margin of the sixth rib. The nipple occupies the center of the area, being either in the fourth interspace or, as I have more often observed it, over the fourth rib. The *mammary line* passes vertically through it and bisects the clavicle at its middle point. In mature females the nipple is displaced through many causes and even in the male it lacks constancy, hence the line is better named the *midclavicular* line. A second vertical line parallel with the mammary line and lying within that line is useful for descriptive purposes, it is called the *parasternal* line and divides the space between the nipple or midclavicular line and the edge of the sternum into equal parts. The *inframammary region* extends from the sixth rib to the lower costal margin. The *sternal region* is bounded by that bone. It should be noted that the length of the sternum is subject to considerable variation and is relatively shorter in women than in men. The transverse projection which marks the union of its first and second parts always can be felt and corresponds with the cartilage of the second rib. The sternal region is divided into upper and lower by the line which separates the infraclavicular space from the mammary region (third rib).

The *axillary regions* meet the mammary and inframammary regions in front; behind they are limited by a vertical line dropped from the posterior axillary fold. The same circumferential line (sixth rib) which divides the mammary regions separates the superior axil-

lary area from the inferior, which last region extends downward to the edge of the ribs. It is convenient to divide these regions by a vertical line, the *midaxillary line,* into equal parts, the anterior axillary region and the posterior region.

The posterior aspect of the chest presents above, the ***scapular regions*** which are anatomically divided into the *supraspinous fossæ* and the *infraspinous fossæ.* The parts included between the inner edges of these bones when the arms are pendant is the ***interscapular region.*** The scapulæ lie on the ribs from the second to the seventh, inclusive. The portion of the chest included between the lower edge of the ribs and a line joining the inferior angles of the scapulæ is the *infrascapular* or *subscapular region.* As an aid to description the regions are bisected by a vertical line called the ***midscapular line,*** which name indicates clearly its location.

Landmarks. The top of the sternum is on a level with the second dorsal vertebra. There is little or no lung behind the first bone of the sternum. The highest part of the aortic arch lies one inch below the suprasternal notch. The innominate artery lies behind and rises as high as the right sterno-clavicular joint. When either the aorta or the innominate is higher than normal its pulsations can be felt in the sternal notch.

No little difficulty may be experienced in counting the ribs in fat persons, hence it is useful to know that:

1.—The transverse projection on the sternum, above referred to, corresponds to the center of the cartilage of the second rib.

2.—The nipple in the male is placed either over the fourth rib, or between the fourth and the fifth ribs three-fourths of an inch outside of their cartilage.

3.—When the arm hangs at the side the lower external border of the pectoralis major corresponds with the position and direction of the fifth rib.

4.—A horizontal line drawn around the chest through the nipples cuts the sixth intercostal space midway between the sternum and the spine, i. e., in the midaxillary line, which point is often selected for tapping pleuritic effusions.

5.—When the arm is raised the highest visible digitation of the serratus magnus corresponds with the sixth rib. The digitations below correspond respectively with the seventh and eighth ribs.

6.—The lower angle of the scapula corresponds to the seventh rib, which is the longest rib in the body.

7.—The eleventh and twelfth ribs can be felt even in corpulent persons.

(These seven points are taken with modifications from Keene's surgical landmarks.)

The tips of the spinous processes of the vertebræ may be made to show readily as red dots by making slight friction over their points.

THORACIC REGIONS AND THEIR CONTENTS.

The supraclavicular region contains two structures of importance to the medical man, the apex of the lung and the subclavian vessels. The apex projects above the clavicle behind the sterno-cleido-mastoid and between the scalenii muscles for a distance varying from one-half an inch to an inch and three-quarters, (Holden). In extreme cases it projects above the rib as much as two inches, especially the left apex which is generally the higher of the two. The artery crosses the first rib at the outer border of the sterno-mastoid muscle where it arches over the lung apex. By elevating the shoulder to relax the tissues the beating of the artery can be felt easily in the subclavian fossa. Just in front of it empties the external jugular vein; behind it the internal jugular.

The *Clavicular region* lies behind the clavicle. Owing to the slant of the ribs the inner third of the clavicle overlies the first rib, the middle third overlies the first and the second ribs. The bone is so separated from the lung that no typical lung note is obtainable, yet direct percussion upon the clavicle, as explained further on, often elicits the first signs of apical change.

The innominate artery extends upwards as high as the upper limit of the right sterno-clavicular joint where it bifurcates into the right common carotid and right subclavian arteries (Morris). The right innominate vein lies immediately outside of the artery. The left common carotid artery lies behind the left sterno-clavicular articulation and the *left* innominate vein passes in front of both innominate and carotid arteries as it traverses the space behind the manubrium to reach the right vein behind the first rib. Between the sterno-clavicular articulations lies the *Suprasternal region*, or *Interclavicular* notch, whose lower border is the top of the sternum. It contains the trachea in the center. The vessels lying behind its joints have been

named. In case of dilatation of the aorta or upward displacement of the heart, the vessel may be elevated above the notch where its pulsations can be felt or even seen. In these cases backward displacement of the trachea is usual.

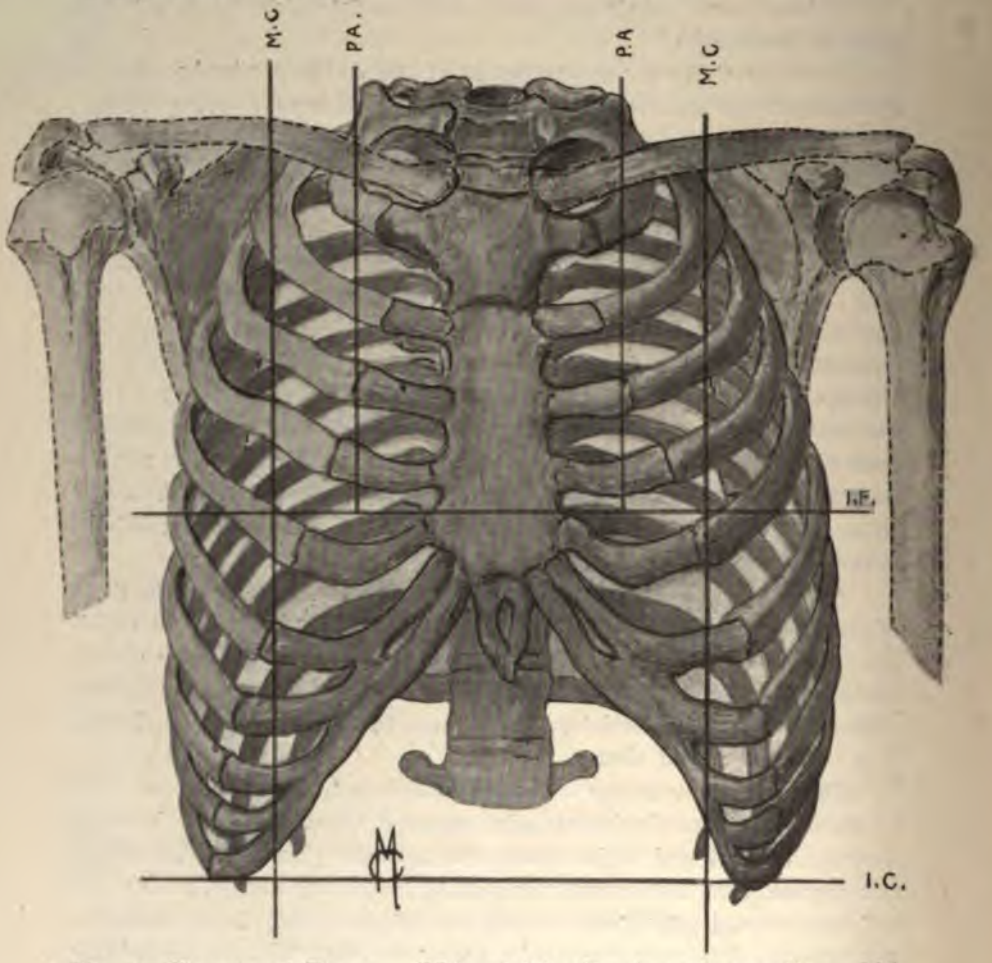


Fig. 10—The normal Thorax. (Male adult.) Showing reference lines. MC Midclavicular line. PA Parasternal line. IF Inframammary line. IC Infracostal line.

The *infraclavicular spaces* overlie the lung substance and as no other structures are in contact with the chest wall within their areas, we get here the typical lung sounds. The superior vena cava lies

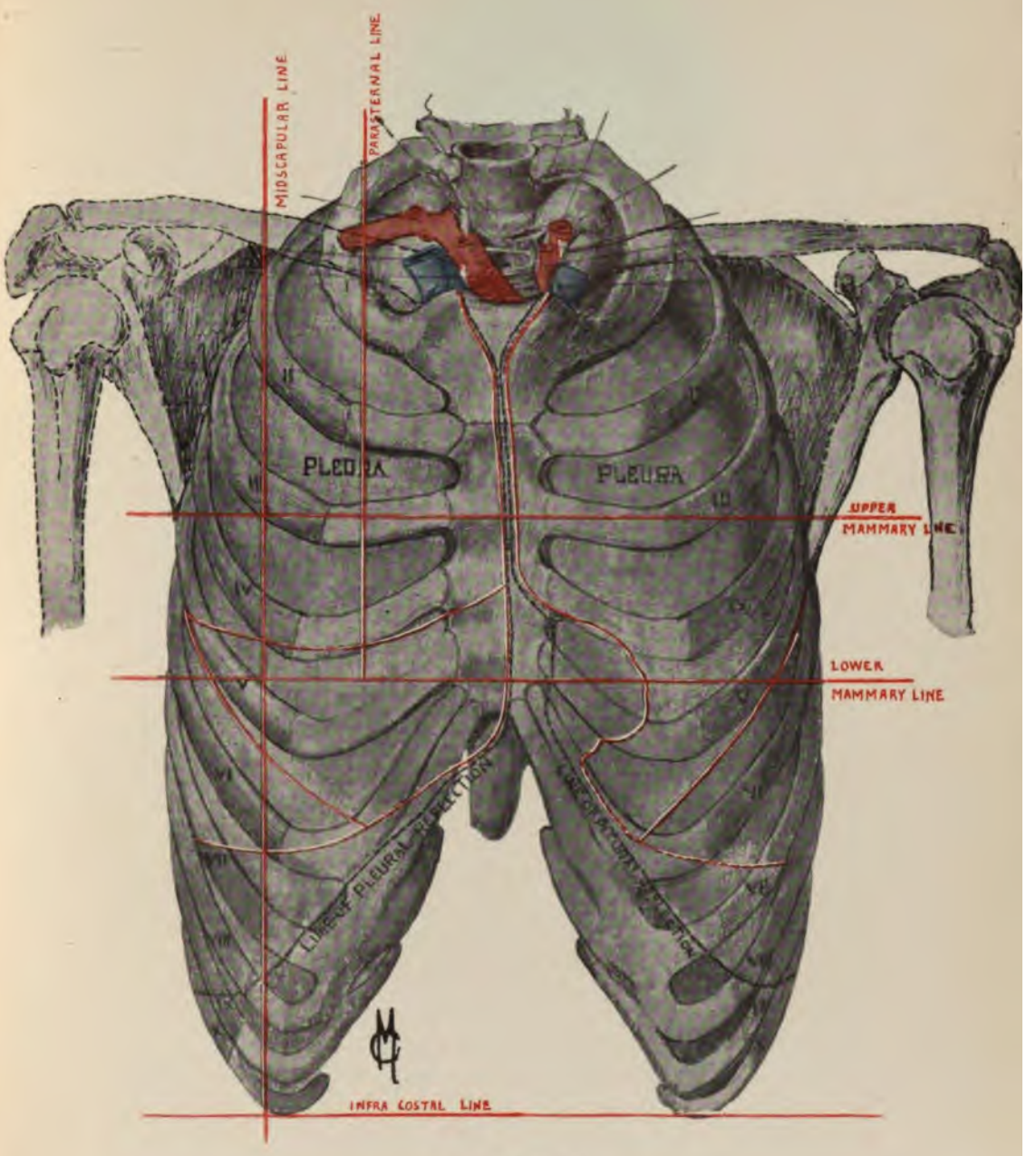


PLATE II.

RELATION OF LUNGS TO THE THORAX

The lobes are outlined in red. The pleural reflection in black. Note the continuation of pleura below lung border.

partly behind the chondro-sternal junction on the right side and behind the first and second intercostal spaces, reaching the right auricle behind the third cartilage (Morris). It is covered by the edge of the lung. The right auricle lies behind the third cartilage and in the third interspace beyond the sternum, but is well covered by the lappet of the right lung.

The *Mammary regions* differ on the two sides. On the right side are lung, liver and heart. The entire right surface area is covered by lung. Beneath it lies the right border of the heart which projects beyond the sternum more than one inch, almost reaching the parasternal line. This border lies behind the third and fourth ribs and their interspaces, covered by the edge of the lung (Luschka). A minute portion of the right ventricle lies in the fifth interspace close to the sternum, which, during forced expiration is uncovered. The liver rises beneath the dome of the diaphragm as high as the fourth interspace, receding farther from the chest wall as it rises.

On the left side the mammary region contains heart and lung. From the left edge of the sternum outward are the right and left ventricles. The left auricular appendix, at the base of the pulmonary artery is quite superficial in the third interspace. The external boundary of the heart on the left side is a line drawn from the point where the parasternal line cuts the third interspace, to a point slightly outside the apex in the fifth interspace, the line curving outward and downward in its direction.

All of this area is covered by lung except the tongue-shaped portion of the ventricle which is known as the area of absolute cardiac dullness, a small triangle whose sides are drawn through these three points, (a) the apex, (b) the base of the xiphoid, and (c) the junction of the fourth rib with the sternum.

The Inframammary region, right side. The lower edge of the right lung follows closely the sixth rib as far out as the nipple line, thence it passes slightly downward and outward to the seventh rib. The diaphragm rises to its highest point just inside the right nipple (midclavicular) line which is as high as the top of the fifth rib (Morris), and separates the lung from the liver. The liver in this same line extends downward to the edge of the ribs where it can be felt on inspiration. On deep inspiration its border projects still lower. This border may be traced upwards towards the umbilicus on an oblique line. In front in the middle line the lower border of the liver extends

downward to a point about half way between the xiphoid cartilage and the navel (Quain). My observation is that this distance is too great, probably one-third being more nearly accurate than one-half, but the distance from the navel to the xiphoid varies greatly. For women the measurement given by Quain is much more nearly correct. Here the line of liver border is from the ninth right to the eighth left costal cartilage, "crossing the middle line about a hand's breadth below the sterno-xiphoid articulation (Godlee).

In order to complete the description of the liver it is only necessary to add that posteriorly it is opposite the ninth, tenth and eleventh dorsal vertebræ, and that on the right side it extends between the seventh and eleventh ribs, and in the mammary line from the fifth to the ninth costal cartilage.

The Left Inframammary region differs materially from the right. The left lobe of the liver extends across the sternum. Its extreme left point is about an inch and a half beyond the left margin of the bone (Quain). The lower lobe of the left lung covers the area, reaching from the fifth to the seventh costal cartilage, then passing outward and downward. The cardiac half of the stomach rises under the left dome of the diaphragm to the top of the fifth rib and extends outward as far as the anterior axillary line. The degree of distention and the nature of the stomach contents influence materially its position.

The Sternal region is divided into upper and lower areas. In inspiration the top of the sternum corresponds to the fibro-cartilage between the second and third thoracic vertebræ and is distant about two and a half inches from the spine (Holden). There is little or no lung behind the first bone of the sternum. The trachea continues downward in the central line and bifurcates behind the joint formed by the manubrium and the gladiolus. Lying on this bifurcation is the aorta, reaching within one inch of the sternal notch. Behind the trachea is the œsophagus. The left innominate vein crosses immediately behind the upper border of the manubrium to reach the superior cava on the right side in the first interspace. Behind the vein ascend the great branches from the aorta. The lower sternal region is the part covered by the gladiolus, or second piece of the bone. Behind its upper area the thin edges of the lungs meet on full inspiration as far down as the fourth rib where the left lung diverges to uncover the heart; the right sometimes extends just beyond the mid-

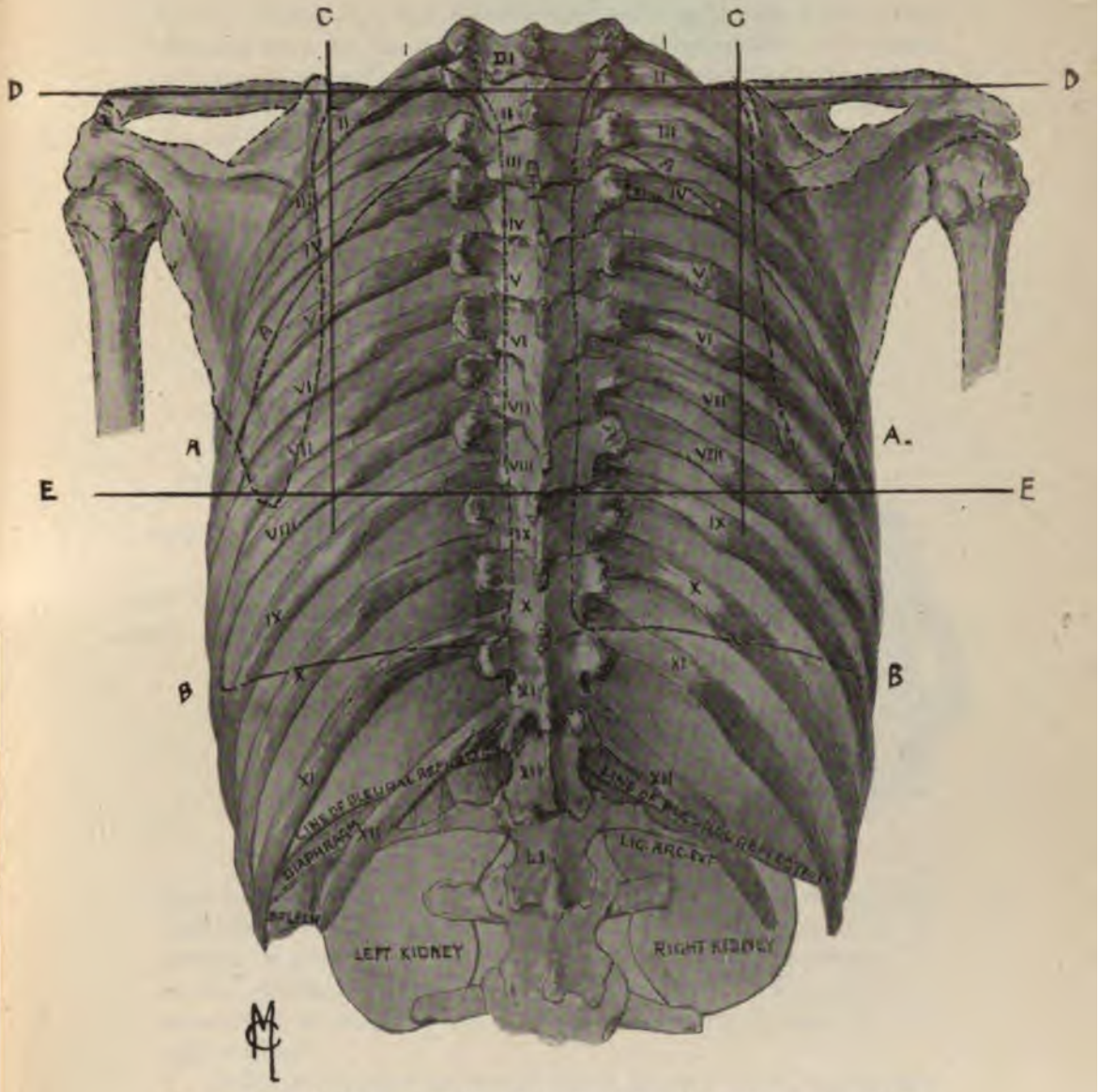


Fig. 11—The Thorax. Posterior Reference Lines. Scapulæ drawn outward. A Line of division between the lobes of the lung. B Lower limit of lung. CC Interscapular lines. D Suprascapular line. E Infrascapular line.

dle line (Holden). The bronchi subdivide outside the edge of the sternum, deep in the mediastinum. The primary bronchi diverge opposite the second rib cartilage. The right is more horizontal than the left and slopes down to the level of the fourth dorsal vertebra. The left is smaller than the right but longer and more inclined, reaching the level of the fifth vertebra. The beginning of the aorta, the

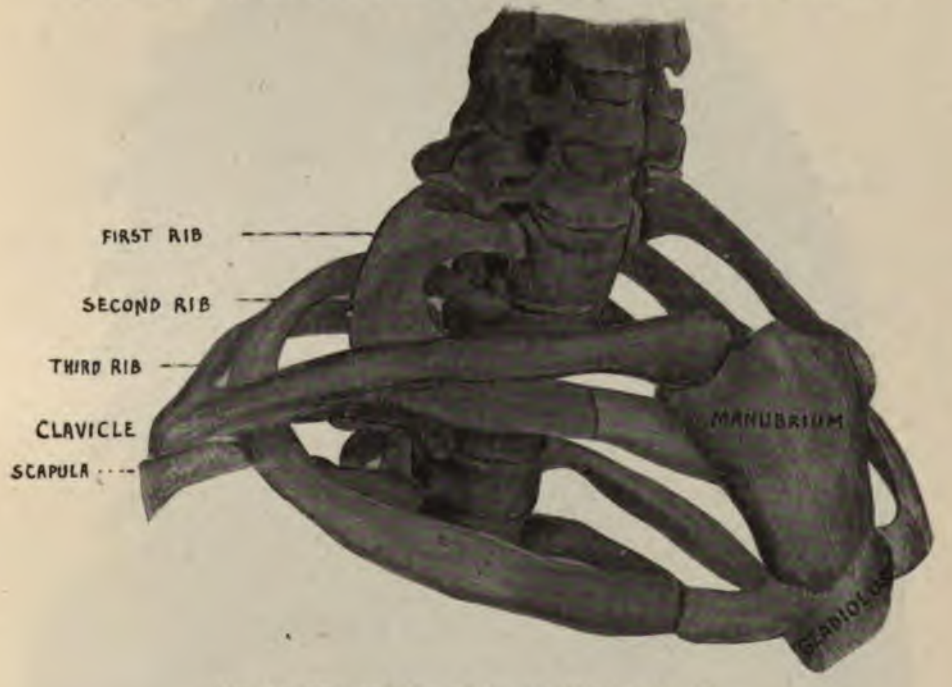


Fig. 12—The relation of the clavicle to the ribs.

inner portion of the right auricle and the central portion of the right ventricle lie to the front in this region. The lower extremity of the sternum is marked by the junction of the ensiform cartilage. It usually recedes from the surface presenting the depression known as the *scrobiculus cordis*, or pit of the stomach, lying opposite the seventh cartilage.

Immediately below the sternal region between the inframammary regions lies a portion of the *Epigastric region*. It is chiefly occupied by stomach and liver. The quadrate lobe of the liver lies immediately to the right of the umbilical line, and adjoining it still further to the right is the gall bladder, opposite the ninth costal cartilage, close to

the outer edge of the rectus muscle. When full the bladder is palpable; when distended its position is often marked by a spherical swelling of the parietes.

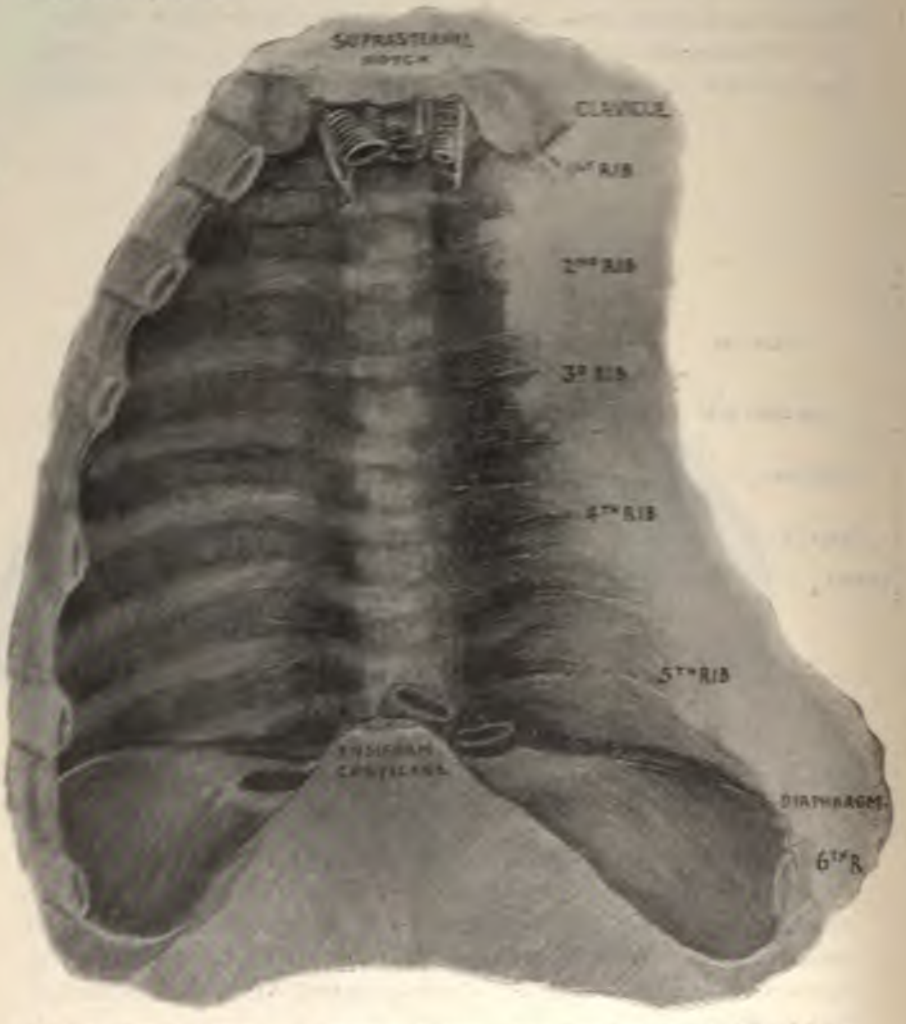


Fig. 13—The diaphragm. Relation to thorax. The caval opening is seen on the left, the œsophageal opening on the right of the ensiform; above it the aorta.

The *Axillary regions* are occupied by lung substance and are alike on the two sides. The lower, or infra-axillary, regions differ as to their contents. On the right side in the posterior axillary line the

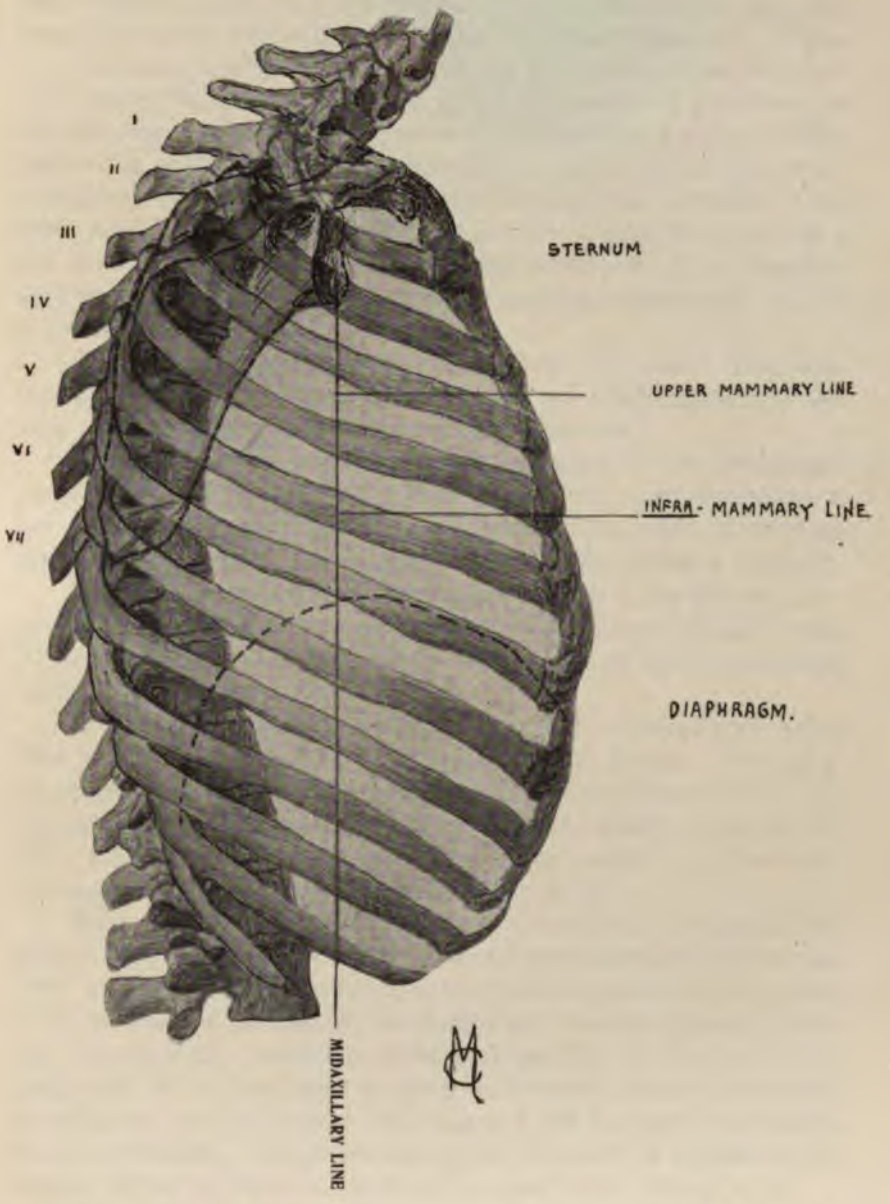


Fig. 14—The normal thorax, with reference lines and position of diaphragm. Lateral view.

lung reaches the eighth rib (Morris). This margin of the lung descends about an inch and a half on full inspiration (Godlee). Hence the numerous discrepancies met with in description. The liver extends below the lower lung margin to the free border of the ribs. On the left side the lung has the same relations as on the right. The spleen lies a little behind the mid-axillary line, its long axis corresponding with the tenth rib, the viscus reaching from the ninth to the lower border of the eleventh rib. Its anterior border is marked by a line drawn from the left sterno-clavicular joint to the tip of the eleventh rib. The cardiac end of the dilated stomach projects into the left infra-axillary region.

The *Scapular regions* do not differ on the two sides. They contain lung tissue. The incisura of upper and middle lobes cuts the fifth rib from above downward on a long diagonal.

The *Interscapular region* is chiefly occupied by the dorsal vertebræ, in front of which is the œsophagus. Still more anteriorly is the trachea with its subdivisions as noted. The point of tracheal division corresponds to the fourth or fifth thoracic vertebræ (Morris).

The *Subscapular region,* from the seventh rib to the inferior margin of the thorax, for the most part covers the lungs. Holden says, "Opposite the angles of the scapulæ, the arms being close to the sides, the lungs extend to the tenth ribs."

It should be emphasized that the pleural *sac* extends lower down than the lung all along the lower margin of the thorax. Thus, near the sternum the lower margin of the sac stretches along the seventh rib cartilage; in the axillary line it reaches the lower margin of the ninth rib; posteriorly it reaches as low as the twelfth rib, which corresponds to the tip of the eleventh thoracic spine.

Holden calls attention to the ease with which tapping of the pleural sac may be performed between the eleventh and twelfth ribs, "but not with a trochar, since a trochar would penetrate both layers of the pleura and go through the diaphragm into the abdominal cavity. The operation should be done cautiously by an incision beginning about two inches from the spine, on the outer border of the erector spinæ on a level between the spines of the eleventh and twelfth thoracic vertebræ. The intercostal artery will not be injured if the opening be made below the middle of the space, which is very wide."

The vertical field of the kidneys corresponds to the last thoracic and the two upper lumbar vertebræ, the right lying in most cases

from a third to a half inch lower than the left, but exceptions to this rule are frequent. They extend inward far enough to overlap the tips of the transverse processes of the vertebræ named.

The spleen slightly overlaps the left kidney opposite the upper half of the kidney's outer border. If the *hiatus diaphragmaticus* is well marked the kidneys come into relation with the pleura, the diaphragm intervening.

The cardiac orifice of the stomach lies to the left of the body of the ninth dorsal vertebra (Holden).

SECTION III.

PHYSICAL EXAMINATION.

INSPECTION.

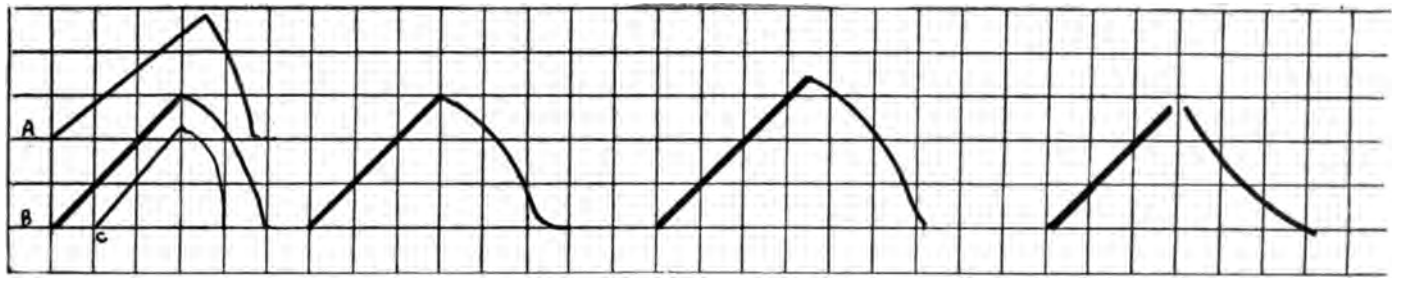
Inspection is the art of observation. Minute differences which the novice utterly overlooks may be detected readily by the trained eye, and valuable information gained thereby. Indeed, it is often possible merely by inspecting the chest to make a near approach to a correct diagnosis. By inspection we recognize changes in size, form and symmetry. In order to give full weight to inspection it is necessary that the chest be uncovered to as great an extent as may be possible without undue risk to the patient by reason of exposure, or unduly wounding the modesty of young females. Place the patient in the best obtainable light; when the condition of the patient admits it is preferable that he stand or sit, since the recumbent posture is not so favorable for observation.

The first point to be observed is the type of the chest, whether long and narrow, flat and shallow, or deep. The normal chest is symmetrical and the diameters well-proportioned. The interspaces are equal and the clavicles none too prominent. Note especially the antero-posterior diameter and compare it with the transverse diameter; carefully compare the two sides as to symmetry or asymmetry, giving especial attention to the anterior, upper surface. Depression here may indicate tuberculous deposits; local bulging in this region may be indicative of aneurysm. Bulging in one lateral area may be caused by a pleural effusion, while retraction of some part may have resulted from previous disease of the lung or pleura. Note anteriorly the outlines of clavicle, sternum and rib, giving attention to inequalities of the interspaces and the degree of obliquity of the ribs; note whether the type of respiration be costal or abdominal; whether expiration is longer than inspiration; whether the intercostal spaces are equal and well-marked; whether the impact of the apex of the heart is perceptible. (Displacement of the heart from various causes is discussed under diseases of that organ. Uncovering of its anterior sur-

face by retraction of the upper lobe of the left lung is referred to under Tuberculosis.) Being familiar with the movements of the healthy chest, you will note changes in the rhythm, frequency and force of both heart and lung movement. All the above observations should be made during quiet, natural breathing; whereupon the patient should be instructed to essay forcible inspiration and expiration, during which the entire category should be repeated, and careful comparison made with the former set of observations. During the period of forced inspiration note carefully the expansion of the thoracic walls, whether it be a uniform, expansile effort, or shows irregularities, as occur in phthisis and after pleurisies; or whether it be a cage-like rise and fall of the entire chest structure, with little or no true expansion, as in emphysema. On the posterior aspect of the chest note whether there are any deviations from the straight line of the vertebral spines; the distance of the inner borders of the scapula from the spine; whether the lower angles are on corresponding levels on the two sides; and whether both or either of these bones project unduly from the body. A valuable sign of pneumonia in children is the lack of expansion just beneath the clavicle on the affected side.

Three types of breathing are recognized: abdominal, superior costal, and inferior costal. In women, as the heritage of the corset, superior costal breathing is the usual type. In men, abdominal. In the normal chest the expansion is equal on the two sides, and all the thoracic diameters increase with inspiration. In disease the expansion may be unequal. If the thoracic walls become fixed, the expansion is chiefly affected by the descent of the diaphragm. It is to be remembered that few persons are perfectly symmetrical; usually in right-handed persons the right side is more developed than the left, while in left-handed persons the opposite obtains. A corresponding slight deviation of the spine is very common and cannot be considered as unnatural. A considerable degree of alteration in the form, movements and size of the chest may occur without serious embarrassment to the contained organs and without interfering with their healthy functions. Few of us have not been struck by the extent to which the ravages of disease may affect an organ, and yet it be able to perform its allotted duty.

Alterations in Chest Walls Produced by Various Diseases. By inspection alone we may detect the presence of various diseases, or conclude that the patient has been affected by them at some more or less

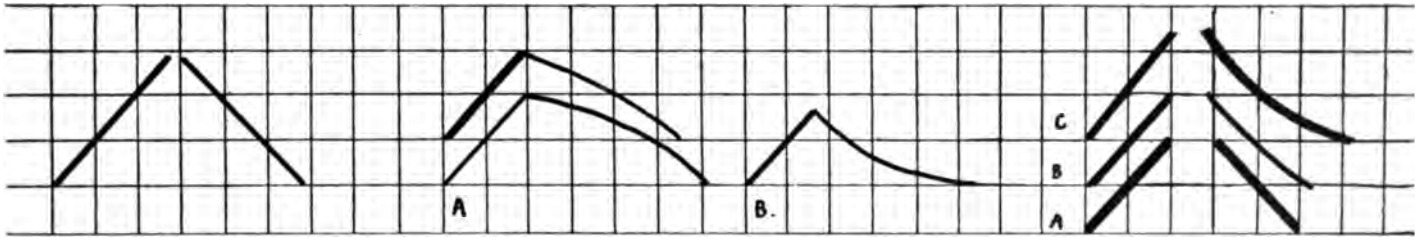


A and B—Normal vesicular breathing; intensity, pitch and length of expiration less than inspiration. C—Shallow breathing; expiration a mere indistinct whiff; silent interval follows expiration.

Purile Breathing. Intensity increased; rate and pitch unchanged; expiration slightly prolonged.

Conscious Breathing. Respirations deeper, i. e., intensity of both inspiration and expiration increased; rate lessened; relative pitch unchanged.

Broncho-Vesicular Breathing. Expiration slightly longer, higher pitched, but less intensity than inspiration—the two separated by a distinct interval.



Broncho-Vesicular Breathing. Inspiration and expiration almost equal in all respects; separated by a short silent interval.

Types of Asthma and Emphysema.
A. Pitch of expiration lowered.
B. Pitch of expiration raised.

Three Types of Bronchial Breathing, inspiration and expiration, separated by a considerable interval. A and B—Ordinary types. C—Exaggerated type.

Fig. 15—Graphic representation of various types of breathing drawn to scale. Each square represents ½ second.

remote period. Thus, pleurisy often leaves an indelible stamp upon its victim. In the pre-effusion stage, and even when only slight effusion is present, there is no apparent change in the size of the chest, but the movements on the affected side will be somewhat restricted and those of the sound side proportionately increased. In the lesser degrees it is difficult to detect this change by the unaided eye. The respirations are not full and free but terminate abruptly, with a catch, as it were, by reason of the pain which develops when a certain degree of expansion is reached. This sign is not distinctive of pleurisy, as it is often present in herpes zoster and intercostal neuralgias.

Irregularities and contractions show the ravages of previous attacks of pleurisy. Old sinuses may exist. Flattened areas, exaggerated interspaces and over prominent fossæ may be the result of tuberculosis. A conical sweling in the upper area may be the first intimation of an aneurysm of the aorta.

Mensuration. Mensuration, as applied to diseases of the chest, furnishes certain data as to shape, size and capacity. By its means the actual outline of the chest is ascertained; the relative measurement of the two sides, a comparison of its various diameters, and the amount of expansion are determined and may be recorded.

The instrument used for determining the outlines of the thorax is the stethometer of Quam, or the cyrtometer. The latter consists of two thin flat strips of lead, each about twenty-four inches in length, joined together at one end by a piece of rubber tubing. The joint is applied over the spine of a vertebra at the desired height, and the flexible strip accurately molded to the chest walls, noting the points where they overlap by a scratch on the bands made over the middle of the sternum. The pieces are removed without disjointing, and a tracing made on a large sheet of paper. The antero-posterior diameter is marked from the joint to the line of overlapping, or may be measured by calipers. The semi-circumferences, the transverse diameters, and other measurements are made from the tracing. Deviations in the two sides are strikingly brought out by superimposing the tracings, which may be done by folding the paper and holding it towards the light. The semi-circumference increases in cases of intra-thoracic growths, or during the accumulation of pleuritic effusion. It decreases in the course of pulmonary tuberculosis, in the deformity following the removal of fluid, and from many other causes.

For ascertaining the relative expansion of the two sides, tapes joined at the back serve an excellent purpose.

Gain or loss of flesh and muscle affect the chest measurements, a fact which is to be taken into account during treatment to re-establish pulmonary expansion, and it must not be forgotten that in right-handed individuals the right semi-circumference of the thorax may exceed the left by as much as an inch.

For ascertaining the comparative outlines of the two anterior surfaces of the thorax, and for making records of the same, I have used with much satisfaction thin, narrow lead strips. The strip is applied vertically so that one end projects over the clavicle, and extends downward to the edge of the thorax. Its relation to the nipple being noted, it is carefully molded over the clavicle and ribs. A tracing of each side is then made. Differences imperceptible to the naked eye are brought out, but what is more important, future changes are detected. The method is especially valuable in incipient phthisis.

Palpation. Palpation is an endeavor to obtain information of the physical condition through the medium of the sense of touch. It consists of laying on the hands, or in bringing the finger tips into contact with various regions of the body. In localized diseased conditions the tips of the fingers should be applied, as the larger area of the palmar surface is not so suitable for differentiating between circumscribed localities, and is less sensitive than the tips. As a means of palpation between the ribs, the ulnar surface of the hand affords an excellent means, owing to its high sensitiveness and the readiness with which it fits into the intercostal depressions.

When the hands are brought in contact with the thoracic wall overlying the lung, and the patient made to speak, a distinct succession of vibrations is communicated to the hand. This is known as *tactile fremitus.** The strength of the vibrations varies in both health and disease from the faintest tremulousness to strong well-marked, separated vibrations. In health the character of the voice materially influences their strength, the vibrations being better marked in low-pitched, deep-voiced men than in higher voices; stronger in men generally than in women, and weakest in children. The amount of tissue overlying the lungs influences this fremitus, and the vibrations are more appreciable in thin persons than in fat

*The phenomenon is indiscriminately called by some writers tactile fremitus and vocal fremitus.

ones. Any form of disease which increases the density of the pulmonary tissue or increases its conduction power augments the fremitus, *provided* that the bronchi are open, and that the air column from the larynx to the pulmonary tissue is uninterrupted. The sensation is due to vibration communicated to the thoracic walls, through the medium of the tissues, from the air in the trachea set in motion by the act of speaking. Hence, fremitus is increased in pneumonia

Fig. 16—Ulnar palpation.

and tubercular deposits. On the other hand, conditions which separate the pulmonary tissues from the chest wall, or diseases accompanied by a diminution of the normal pulmonary density, lessen the fremitus, or even obliterate it. Thus, when a layer of air or fluid intervenes between the periphery of the lung and the parietal wall, as occurs in pleuritic effusions, plastic pleuritic thickening, pneumothorax and in solid tumors the fremitus is decidedly lessened or is totally absent. In health the fremitus is most marked on the right side just below the clavicle where it is stronger than in the corre-

sponding left area, probably due to the anatomical fact that the right bronchus is of larger caliber, is straighter and enters the lung on a somewhat higher level than its fellow. Posteriorly, in the right supra-scapular fossa the fremitus is greater than on the opposite side, for the same reason. From this fact it may be deduced that if the tactile fremitus is well-marked and *equal* on the two sides in the areas named, then it is *increased* on the left side, and further evidences of disease should be sought for at the left apex. If fremitus is weakened but equal on the two sides it would indicate *diminution* on the right side.

Fig. 17—Differential palpation of apices of lungs.

When the bronchial tubes are more or less occluded by the adherence of tenacious secretions or by stenosis, vibrations similar in character to the voice vibrations, produced by the passage of the air through the bronchi, are communicated to the hand. This is called *rhonchial fremitus.* It is often present in bronchitis, and is particularly frequent in children suffering with the disease. The conditions necessary for its production are the same as those which produce the râles heard on auscultation.

Fremitus produced by coughing is called *tussile.* Advantage may sometimes be conveniently taken of crying spells in young children to determine the presence or absence of crying and tussile fremitus.

Friction fremitus results when two inflamed surfaces of the pleura are rubbed together, and is often present during the dry stage of pleurisy. More rarely it is due to pericardial or peritoneal friction, which however may sometimes be felt.

Next in importance to fremitus as a contribution to our knowl-

edge by palpation ranks the *location of the cardiac apex*, as indicated by the position of the impulse. Even when visible on inspection it may be more accurately located by palpation, and a consideration of the many diseases which affect its character and location will impress on one's mind the extreme importance attached thereto. Fowler says: "The position of the cardiac impulse is the key to the diagnosis of many affections of the chest."

Other information gained by palpation is a recognition of the degrees of resistance offered by various organs, and especially resistance due to increased density of an organ. The finger-tips may detect the presence of pulsation or vascular thrill, as of an aneurysm, crepitation caused by air under the skin, or crepitus, such as results from a broken rib.

The relative length of the expansile excursion on breathing may be appreciated by many better by palpation than by inspection. Hyperæsthesia and tenderness are revealed by palpation.

THE PULSE.

Much valuable information is gained by a careful study of the pulse, which with many becomes such a routine procedure that the gains are minimized. Intelligent palpation of the pulse enables us to recognize the febrile state, to estimate the general strength of the patient during the course of disease, as well as to suspect solely from its character the presence of various conditions and complications. The three chief factors which are to be noticed in examining the pulse are the *frequency*, the *quality*, the *rhythm*.

The frequency of the pulse is easily ascertained in general although it is possible for it to attain a rate so rapid as to be uncountable. The right radial artery is chosen for convenience and the examiner's left finger tips are lightly superimposed. The count is made during fifteen or thirty seconds, unless great accuracy is desired, when the beats are counted during one or two minutes. The normal pulse rate for healthy adults is usually stated as being seventy-two per minute, but the rate is sometimes higher and often much lower. The pulse is more rapid both in childhood and in old age, and varies much in different individuals of the same age. Eichhorst's figures are given thus, slightly modified for ease in memorizing:—first year, 135 per minute; second year 110, falling to 100 at the fifth year; then falling two beats per year for the next five years, giving us 90 at the

tenth year; reaching 72 between the fifteenth and twentieth year, where the rate remains stationary until the sixtieth year, rising slowly year by year thereafter.

Many conditions aside from disease influence the pulse rate, either increasing or diminishing it. Thus alcoholic stimulants increase it and their persistent use brings about a maintained high pulse rate. Tea and coffee increase both the force and frequency of the pulse, as does the taking of food, while fasting diminishes both. The effects of exercise, excitement and emotions of an exhilarating nature are well known, while depressing emotions, fear and shock diminish its rate, as do rest and sleep. Quiet sleep, recumbency, sitting, standing and moving each successively augment the pulse in the order named. High temperatures and high atmospheric pressure increase, while the opposite conditions diminish the rate. Deep breathing and coughing increase the volume as well as the number of beats. The pulse is influenced by drugs, and it is wise to ask what medicines have been taken before estimating the pulse value. Digitalis is the commonest drug used to slow the heart; belladonna has an opposite effect.

The pulse during the twenty-four hours describes two well-defined cycles which vary greatly in different persons, although showing considerable constancy in the individual. These show a morning and an evening maximum, and an afternoon and a night minimum rate. In incipient phthisis the day cycle is often reversed.

Morbid changes which influence the pulse rate are too many to be enumerated fully, but a few may be mentioned with profit. All inflammatory conditions and fevers raise the rate, and in uncomplicated fevers the pulse rate increases about 8 to 10 beats for each degree F. above the normal. This rule is not applicable to persons who were the subjects of disease prior to the onset of the fever. In such the rate rises in a much higher ratio. The various infective processes, pyæmia, septicæmia, tuberculosis and the like are all attended by rapid pulse. Increased intra-thoracic pressure from almost any cause accelerates the pulse, apart from the inflammatory action which may be the cause of the pressure; thus effusions, obstructions caused by disease of lung or pleura, enlarged glands, so act. In incipient phthisis *the pulse constantly augments long before the temperature* curve shows its characteristic rise. Pain increases the rate; so also does pneumogastric paralysis, as from glandular or aneurysmal pressure. The rapid pulse of exophthalmos is one of the three classic

symptoms of that malady. Most anæmias are attended by rapid pulse. The technical title is *pulsus frequens.*

A diminished pulse rate, while less frequently a manifestation of morbid processes, is perhaps more significant than its opposite. The absorption or presence in the blood of certain substances manufactured within the organism, as urea, bile, bile pigment, etc., slow the pulse. Cardiac degeneration, especially if accompanied by disease of the coronary arteries, some forms of valvular disease and affections of the myocardium, very generally slow the pulse. Of the valve lesions, aortic stenosis stands first in this relation, followed by mitral stenosis. All forms of weakness, especially post-febrile and post-hæmorrhagic debility, influence the rate in the same manner. A large percentage of epileptics, as pointed out by Craig, are the subjects of valvular heart diseases; but aside from this they furnish a pulse rate most striking for its slowness. Wood records a rate of 8 to 10 per minute. All forms of disease of the brain and meninges accompanied by increased intercerebral pressure, retard the pulse. Lead intoxication slows the pulse. Many other conditions will also fall within the experience of the practitioner. The condition is spoken of as *pulsus rarus*, but in extreme slowness the precaution of counting the systolic contractions of the heart always should be observed, as the wave may not reach the extremity.

The quality of the pulse depends upon the balance maintained between its size, its strength and its fullness. The amount of expansion which the artery undergoes depends upon the strength and the fullness. The strength varies with the strength of the cardiac contraction, and the fullness with the amount of fluid pumped into the channels. The latter is much influenced by the state of the venous system. Without material difference in the rate the artery may swell gradually under the finger, or the vessel may fill with unwonted suddenness, to subside as quickly as it arose. Such conditions often point to organic diseases of the valves.

The last described of these conditions, spoken of as the pulse of unfilled arteries, the recedent pulse, or *pulsus celer*, often enables us definitely to recognize aortic incompetency. The peculiarity is made more strikingly manifest if the patient's arms are raised vertically above his head while examining the radials. With this pulse is often seen marked pulsations in the vessels of the neck and elsewhere. It

is called Corrigan's pulse, from its describer. Its sudden, slapping, jerky character has given it the name, "water-hammer pulse."

States of high arterial tension give us a hard, full pulse. The vessel walls are not hard, yet the pulse may have a cord-like feel. The radial pulse may be misleading, as it is sometimes anomalous, and is easily pressed upon by clothing.

We estimate the strength of the pulse by the amount of pressure of the fingers necessary to obliterate it. A really weak pulse is accompanied by a diminution in intensity of the first sound of the heart, without much change in the intensity of the second sound. Obstruction in front as well as increase of the *vis-a-tergo* give us a hard pulse, hence mitral stenosis, aortic insufficiency, contracted kidneys, hepatic cirrhosis and many other conditions produce it. The distinction between a hard pulse and hardness of the arterial coats due to sclerosis, should be borne in mind. The differentiation is simple.

Pulse Rhythm. In health the pulse is perfectly regular. However, during sleep, especially in children, intermissions may occur which are in no wise due to deleterious influences. The study of the pulse often shows a distinct intermission recurring at regular intervals, as the loss of a beat at every fourth, sixth or eighth contraction; or the intermission may be irregular. The pulse may be affected without the heart being correspondingly involved, or the heart beat also may be abortive. Sometimes several slow pulse beats are followed by two or three rapid ones. The significance varies greatly. Excessive use of tobacco is one of the most common causes of arythmic pulse. Arhythmia is sometimes a premonition of oncoming cerebral disturbances. It is common in pericarditis, especially of children, and in many forms of heart disease, particularly fatty heart, of which it is, perhaps, the most pronounced sign, of mitral disease and various functional disturbances, as flatulency or dyspepsia. It is prone to occur during convalescence from debilitating diseases, where its significance is less ominous than its occurrence during the height of the ailment, when it often portends heart failure.

The pulse of the two sides may show want of symmetry, as often occurs in aneurysm of the arch of the aorta causing obstruction of some of the branches. Owing to the same cause the two pulses may not be perfectly synchronous. Embolism may cause diminution or obliteration of the pulse, but in such cases a careful search for anomalies should be made.

THRILL.

In mitral and aortic disease a distinct thrill is sometimes imparted to the pulse, and should always suggest the causative factor.

Dicrotous Pulse and *Pulsus Bigeminous.* Sometimes a curious double beat may be felt by applying the fingers very lightly over the artery. It is not uncommon during convalescence from fevers, especially typhoid. When the two beats follow each other closely, it is called "bigeminous"; generally the second beat is weaker. The sphygmograph more clearly shows these peculiarities. Its significance is not particularly important.

VASCULAR PHENOMEINA.

A study of the various phenomena exhibited by the veins and arteries is both interesting and profitable although much neglected by the student in general.

The veins from the examination of which may be gained most information are those of the neck, especially the internal and external jugulars, although the superficial veins of the chest, abdomen, legs and other parts are capable of imparting many supplemental facts. The phenomena presented are studied by inspection, palpation, and auscultation.

I. *Venous Pulsation.* On inspection there is often present a visible throbbing in the veins of the neck, the internal and external jugulars being especially the seat of pulsation. By reason of the direct connection with the superior vena cava, the right internal jugular is more apt to show pulsation than its fellow. The significance of the venous throb in the neck has been referred to under cardiac inspection. The venæ innominatæ or brachio-cephalic sinuses at the root of the neck, just behind the clavicle, should be carefully examined. Palpation may reveal pulsations which are invisible.

II. *Enlargement of the veins* is of common occurrence and may be temporary, permanent, variable or intermittent. Kinking, knotting and varicosities are present. They are far more common in women than in men, and become more apparent with the advance of age. Permanent enlargement of the veins results from dilatation or continued engorgement of the right heart, from tricuspid regurgitation, from obstruction affecting a large venous channel as the cava. Such obstruction may be caused by extreme pressure from an enlarged organ, as occurs in hepatic cirrhosis, by a new growth, such as a tumor

or an aneurysm, or from occlusion of the vascular lumen by a thrombus or an embolus. The enlargement may be due to the communication of an aneurysm with the superior cava, of which accident Pepper and Griffith have collected twenty-nine cases.. "Cyanosis, œdema and great distention of the veins of the upper part of the body, being the most frequent symptoms."

Thrombosis of the portal vein may occur as a sequence of cirrhosis or syphilis of the liver, or be caused by cancer or by sclerosis of the vein itself. The condition is known as adhesive pylephlebitis. Intense engorgement of the entire portal system rapidly follows. The superficial abdominal veins are distended and tortuous. Hæmatemesis, melæna, ascites and enlargement of liver and spleen give intimations of the condition. A definite diagnosis is rarely reached during life.

The distention of the cervical veins during cough is a familiar spectacle, but during the course of diseases which permanently enlarge the veins, and thus produce insufficiency of the valves, the swelling is much increased during the act and even may be alarming, as in a recent case of my own of a patient in the terminal stages of phthisis. In extreme cases the pulsation can be both seen and felt. An easy method of determining whether the pulsation is transmitted or is due to filling from *below* is to make just sufficient pressure on the vein to obliterate its lumen at its lowest palpable point in the neck, then to slide the finger upwards for a short distance with only sufficient force to empty the vessel. If the vessel fills from below it does so with a jerk which is synchronous with systole and the carotid pulse. The external jugular offers the best opportunity for the trial.

Venous distention increases during expiration and diminishes during inspiration. As already mentioned, tricuspid regurgitation and right-sided engorgement and hypertrophy are the most potent causes of the phenomena. Insufficiency of the valves within the veins has been discussed. Friedreich's diastolic collapse as a sign of pericardial adhesion is described elsewhere. Venous pulsation is often seen in chorea and chlorosis. The venous engorgement, particularly of the small veins of the thorax, in emphysema is mentioned thereunder.

Most cardiac valvular diseases, especially when accompanied by lack of compensation, produce venous engorgement, of which local and general œdemas are the sequels.

The enlargement of the veins around the umbilicus, known as the

caput medusæ, is due to thrombosis or obliteration of the portal vein, as occurs in cirrhosis of the liver.

In phthisis venous pulsations are common, an occasional situation being the back of the hand.

VENOUS MURMURS.

Auscultation. The "bruit de diable" of French writers, the venous hum of American authors, is often present in health, but is generally accepted as significant of anæmia wherein its intensity furnishes an index of the grade of the anæmia for the particular case; that is to say, that while it varies widely in the different forms of anæmia, and in different individuals, yet in general its intensity lessens with improvement and increases with increase in the malady.

The bruit is best heard at the junction of the right subclavian with the jugular vein, the head being twisted slightly to the opposite side. The murmur is not interrupted as is the arterial murmur, but is continuous. Its quality varies and the names descriptive thereof are as numerous as those chosen to describe valvular lesions. It is usually humming or buzzing, or rustling, but may be musical, singing, piping or whistling. Pressure of the stethoscope produces it in health and pressure intensifies it when otherwise present. It is strongest during inspiration and during cardiac systole. It diminishes and may disappear on lying down and is loudest when the patient is erect. Sometimes a venous thrill is felt in the vessels of the neck in cases of extreme anæmia, but is quite rare.

Time of Venous Pulsations. The venous pulse as observed in the external jugular is either presystolic or systolic. It may be timed by the carotid pulse. The presystolic pulsation is spoken of as the negative pulse. The vein *collapses* with the heart's systole and refills before the next systole. The collapse is synchronous with the apex beat, hence the refilling is synchronous with the auricular contraction which closes diastole and is presystolic.

The systolic venous pulse, spoken of as the positive pulse, is synchronous with the apex beat. It is most apparent in the right internal jugular vein and is a very important and positive sign of tricuspid regurgitation. To a lesser degree other veins are affected and show throbbing. In the form of valvular deficiency just mentioned this pulsation is produced by the systole of the right ventricle projecting a current of blood through the insufficient tricuspid valve

at each contraction, over-distending the already filling auricle and projecting the blood still farther into the ascending cava whose open mouth is unguarded by valves.

A similar blood wave traverses the inferior cava and gives rise to pulsations in the liver produced and explained in the same manner and having the same time as the jugular pulsations. The change in the volume of the organ which occurs with expansion and contraction of the heart may sometimes be distinctly felt and even seen. Ofttimes the throbbings are painful.

The phenomenon is best appreciated by placing one hand over the fifth and sixth interspaces at the junction of the costal cartilages and the other over the liver in the mid-axillary line, when the rhythmic, systolic expansile pulsation is felt easily and can be distinguished readily from the systolic *depression* of the liver caused by communicated pulsations from the hypertrophied right ventricle, which is non-expansile.

ARTERIAL MURMURS.

Through the large arteries the sounds of the heart are transmitted for a considerable distance by the blood current and have the qualities of the sounds as heard at the aortic valves. The intensity diminishes as would be supposed as we recede from the point of origin. In the neck over the seat of the common carotids or the subclavian artery both heart sounds are distinct, but the sound made by the closure of the aortic valve, the cardiac second sound, is the more evident. The first heart sound is here faint and of low pitch.

Over the exit of the femoral arteries from under Poupart's ligament the aortic valve sound may often be distinguished in health, but disappears before Hunter's canal is reached. At the ligament the first heart sound can not be heard.

Arterial Murmurs due to Disease. Just as pressure with a stethoscope upon the trunk of an artery causes a murmur to appear, so pressure arising from various causes as tubercular deposits, new growths, enlarged glands, thyroid tumors or aneurysms occasion arterial murmurs.

Diseases of the vessels which change their caliber or destroy the resiliency of their coverings produce murmurs, hence the aneurysmal bruit and the murmur associated with extensive atheroma. This disease is prone to attack the aorta just beyond the sinuses of Valsalva,

and many a case of supposed aortic valvular disease is in reality due to the bruit or the thrill caused by the calcification plates. Congenital narrowing of the vessels operates in the same way as external pressure to produce bruits. Thrombosis may also cause a murmur. The uterine bruit which accompanies the formation of the placental sinuses during pregnancy is a familiar example of vascular murmur.

In aortic insufficiency the vascular phenomena are characteristic and it is possible to make a diagnosis of the disease by inspection and palpation alone. Thus:

On *Inspection* a visible pulsation is seen in the peripheral vessels, arteries, veins and capillaries, and often the face or hands are seen to alternately suffuse and pale with the heart action. The capillary pulse is seen in the finger nails or along the margins of a light scratch, or by pressing a microscopic slide against the everted mucous membrane of the lower lips, as suggested by Shattuck. The arteries pulsate forcibly, the carotids swell and throb, the aorta behind the suprasternal notch rises and suggests aneurysm, the abdominal aorta lifts the epigastrium with each beat. The temporals and radials fill with a vermicular twist and jerk, followed by instant collapse. The ophthalmoscope shows the same phenomena in the retinals.

Palpation. The finger feels a short, quick, hammer-like stroke, followed by rapid recedence. When timed with the apex beat the radial pulse shows a *distinct retardation.*

Auscultation. In the carotids and subclavians a double murmur is heard, and the second aortic sound may be audible here, even when absent at the aortic cartilage. Duroziez calls attention to the double murmur heard in the femorals.

Tyson mentions a vascular sign described by Traube, occurring in aortic regurgitation as the result of the sudden change of pressure. "This is usually a double sound, of which the first element is ascribed to the rapid distention of the artery by a blood wave which throws its walls into vibration. A second sound occurs with a cessation of the pressure."

Intermittent venous murmurs are spoken of by some authors; they must be of rare occurrence.

THE SPHYGMOGRAPH.

Numerous instruments have been devised for the graphic representation of the pulse in the human being. Marey's sphygmograph

is one of the oldest, but is still most used. Richardson's or Dudgeon's modification of Ponds' American sphygmograph is the simplest form for the clinician. By means of the instrument a graphic record of the pulse is made in the form of a tracing, which shows a saw-like series of elevations and depressions corresponding to the vibrations in the pulse wave.

The elevations correspond to the onset of the wave, the depressions to its recedence. Hence variations in the tracing graphically show differences in the temporary rise and fall of arterial pressure, the rate at which successive waves pass a given point, as well as varia-

Fig. 18—Dudgeon's Sphygmograph.

tions in their rhythm. The primary blood wave, caused by the cardial systole is followed by several succeeding waves of lesser degree, called secondary waves. The chief of these is the *rebound* in the blood current caused by the sudden check, produced by the closure of the aortic valves, named the *dicrotic wave.* A lesser wave, supposed to be due to the stoppage of the backward flow towards the heart of the aortic current, by the closure of the valves, is called the *predicrotic wave.* Perhaps the following explanation will render clear the difference in the two waves. The systole forces the current up hill into the aorta. A portion of the fluid obeys the impulse to return. The gates close. The first influence felt by the column in the arteries is the *stoppage*—the predicrotic wave. The second effect is the *rebound*—the dicrotic wave. This dicrotic wave is increased by the natural resilient recoil of the elastic aortic walls, dilated by the primary current, which occurs just in time to catch the second wave. Lesser waves due to this resiliency follow the dicrotic wave but are of less moment.

The dicrotic *notch* is due to the cessation of the inflow with each systole, causing a momentary falling off of the pressure, the fall being modified by the influences just named.

When the arterioles are constricted, the dicrotic notch is lessened and we have the curve of high tension, pulse such as occurs in Bright's disease. When the arterioles are widely dilated, the dicrotic notch is exaggerated and we have the low tension characteristic curve. Before making the tracing it is well to measure the varying degrees of pulse tension by the sphygmometer. The pressure of the instrument is adjusted accordingly. In pulse of low tension, slight pressure gives the

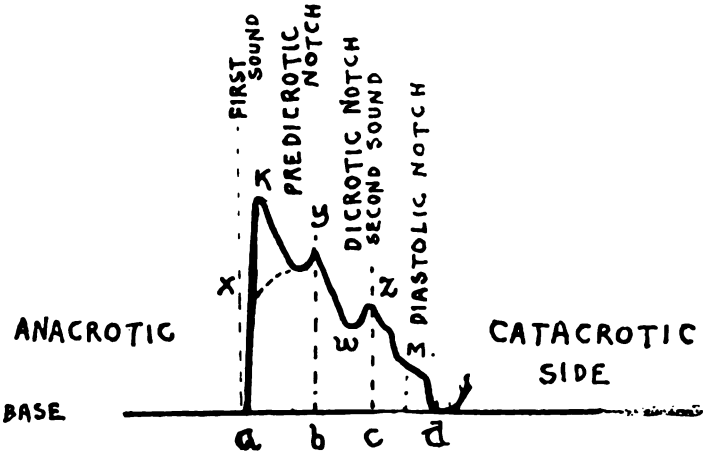


Fig. 19—Normal pulse tracing.

best results and too much pressure extinguishes the tracing. In high tension pulse, rather strong pressure of the spring button gives the best results.

Ax, entrance of the stream into the aorta. This up-stroke is called the anacrotic stroke, or percussion stroke, and is due to the sudden dilatation of the artery by the primary blood wave. The hypersensitive lever is carried too high, and in reality should have stopped at *x*. The lever begins to fall but is overtaken by the predicrotic wave on the catacrotic side of the tracing and carried to *y*—, the *real* summit of the pulse wave. Hence omitting the false movement of the lever, *axy*, represents the real upward or anacrotic curve, *ky*, represents the predicrotic notch, *y*, represents the point at which the ventricular stream ceases, when the positive pressure remits and the negative

wave, caused by the sudden cessation of inflowing blood, and the first slight reflux begins. This cessation of pressure allows the lever to fall to *w*. *W*, is the point of closure of the aortic valves and is *synchronous* with the *second* sound of the heart. *Wz*, is the positive wave or rebound due to the closure of the aortic valves and corresponds to the true dicrotic wave, hence *ywz*, is the dicrotic notch, sometimes called the aortic notch, owing to the manner of its production. The slight curve at *m*, is due to the resilient waves which succeed the aortic or

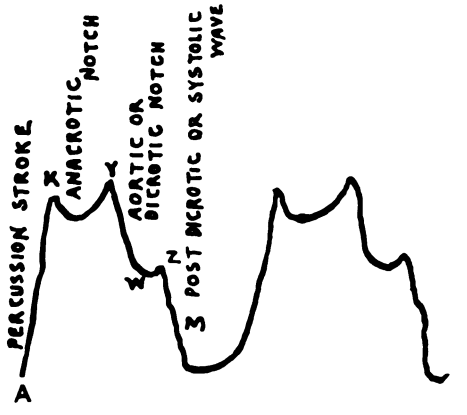


Fig. 20—Pulse of constricted arterioles. High tension pulse.

dicrotic wave. Since this curve *m*, is produced during diastole, it is sometimes caled the diastolic notch.

The Pulse of High Arterial Tension. The pulse of constricted arterioles, the high tension arterial pulse, called by Sansom the pulse of prolonged arterial tension, differs from the normal pulse in that a notch is formed at the top of the curve and the maximum altitude of the lever may be reached *after* the notch. Yeo explains this phenomenon on the ground that the first reflected wave is positive instead of negative, in the case of constricted arterioles, and is therefore added to the percussion stroke, reaching it just as the lever begins to fall at *k* and carrying it to *y*, thus broadening the top of the primary curve, and this pulse is named the anacrotic pulse. The aortic or dicrotic notch, *wz*, is lessened as the dicrotic wave is lessened in these conditions and the post-dicrotic wave *m*, is not discernible. Such a pulse is the result of resistance encountered by the blood current.

through the arterioles and capillaries, as stated, and is found in contracted kidneys, in all diseases which cause atheroma of the arterial walls, gout, lead-poisoning, and aortic stenosis.

Lead-poisoning is prone to produce three of the chief disease-factors just mentioned, since lead workers are notoriously subject to arterio-sclerosis, contracted kidneys and hypertrophy of the heart. In this state the fullness of the vessels is felt to be maintained for a considerable period and the collapse is gradual. Pressure of the finger obliterates the pulse with difficulty.

The Pulse of Low Arterial Tension. In this case the arterioles are widely dilated, the negative wave is reflected and is subtracted from the primary wave instead of being added to it. The vessels collapse

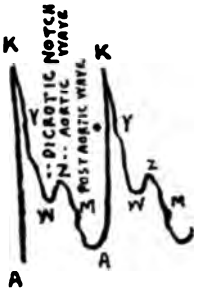


Fig. 21—Pulse of low arterial tension.

quickly after the primary filling and pressure of the finger easily obliterates the pulse. Such a pulse is said to be soft. The tracing shows a vertical up-stroke, *ak*, followed by a quick fall, *ky*, so that the apex, *k*, is sharp and pointed. The predicrotic notch, *y*, is insignificant, but the dicrotic notch, *wz*, and aortic wave, *z*, caused by the valve closure are exaggerated, also the post-aortic curve, *m*, is marked. When present in even a moderate degree the wave, *z*, may be appreciated by palpation and the pulse is said to be dicrotic. Such dicrotism may reach an extreme degree and the point, *w*, may fall to the base line or below, in which case the pulse is said to be hyperdicrotic.

Low tension pulse occurs with conditions which cause dilatation of the capillaries, as the inhalation of chloroform, the administration of nitro-glycerine or nitrite of amyl. Turkish baths are followed by the low tension pulse. In diseases producing serous diarrhœas, colliquative sweats and diuresis it follows, owing to the emptying of the vessels. It often marks the course of febrile diseases. Aortic regurgi-

tation furnishes the best examples. When the dicrotic wave approaches in force to equality with the primary wave, to the finger it appears as though there were two closely associated beats followed by a pause. Such a pulse is called pulsus bigeminus. (See section on Pulse.)

Sometimes three such beats are separated by a pause when the name pulsus trigeminus is used to designate the condition.

THE SPHYGMOMETER.

This is an instrument intended to measure the degree of tension in the peripheral arteries with greater accuracy than the time honored

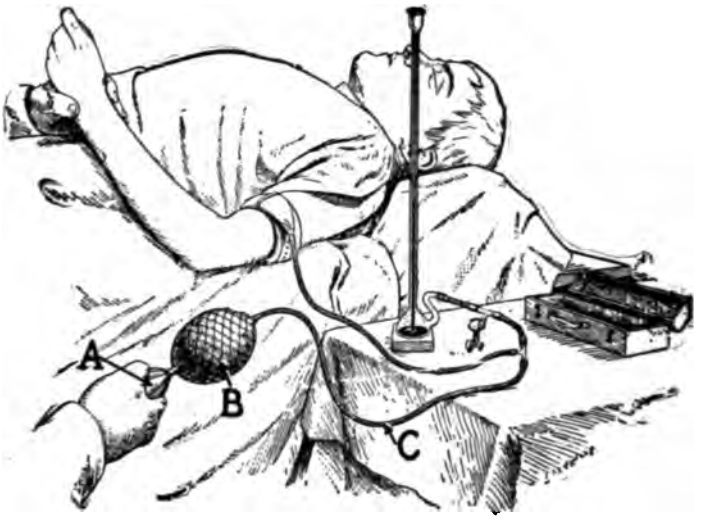


Fig. 22—The Riva Rocci Sphygmomanometer.

one of simple palpation. By its use records can be made of daily variations during the course of disease and operations, as well as the ascertaining of immediate results from taking medicine, food, and drink.

A number of these instruments have been devised, some supposed to measure the systolic pressure of the artery, by recording the amount of force required to obliterate the pulse, others to measure diastolic pressure. The recorders are of two kinds, manometers, or mercurial columns, and aneroid instruments, fitted with dials, like the barometer. The latter are objectionable owing to the readiness with which their

mechanism is disordered. The objection to the instruments in general is that they are provided with rubber tubing which inflates to varying degrees under pressure and changes much in this respect with age.

The Riva-Rocci instrument is, all things considered, the most serviceable. Stanton, of the University of Pennsylvania, has modified

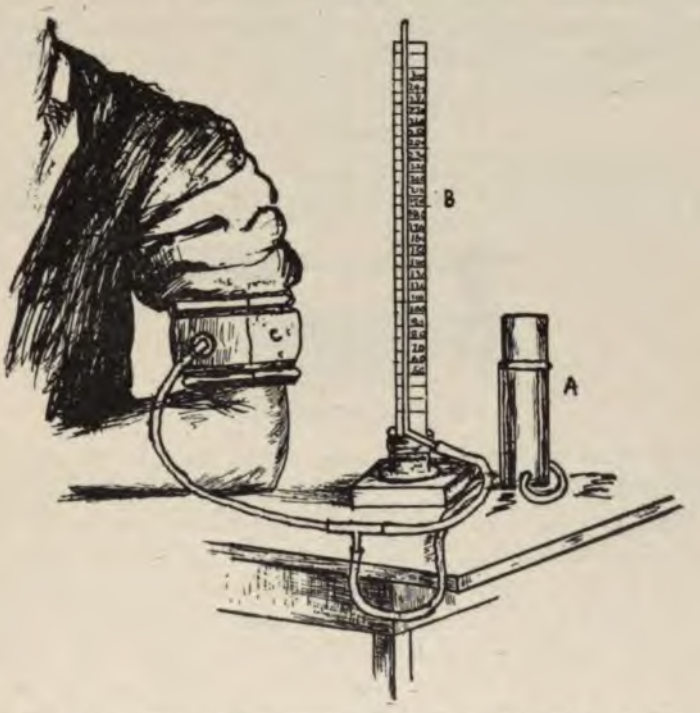


Fig. 23—The Riva-Rocci Sphygmometer—Stanton modification. A Air pump. B Manometer. C Cuff.

this by fitting it with rigid connections. The essential parts are an air pump, a manometer, a rubber armlet which fits around the upper arm, and tubing connections. Stanton advises that the width of the armlet be not less than three and a-quarter inches. To use the instrument the armlet is adjusted, air is pumped in until the radial pulse is exactly extinguished, and the height of the mercury noted at the same moment. Experiments have shown that the pressure within the armlet and that within the manometer are at all times the same. The accuracy of the instrument is shown by applying it to the thigh, when

it will be found that the difference in the index does not vary over four or five millimeters.

The slight oscillations noticed are caused by the varying degree of interarterial pressure and the influence of respiration on blood pressure. During the course of protracted operations the instrument furnishes valuable information, likewise in the study of the state of arterial tension in kidney and heart diseases, and the effect of drugs. It is difficult to transport, is fragile and, like many other devices, has so far found its field of greatest usefulness in the hospital.

PERCUSSION.

Percussion elicits certain sounds by tapping over the areas occupied by the thoracic and abdominal organs. The tapping may be

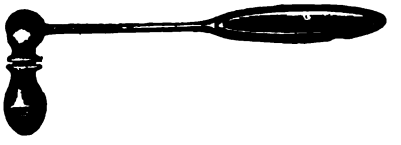


Fig. 24—Percussion Hammer.

directly upon the overlying tissues, called mediate, or upon an imposed substance, immediate. While the rubber-headed hammer named a plessor, and an ivory plate called a pleximeter, are useful for class demonstrations, nothing in actual practice equals the fingers of the two hands, as thereby we not only quite as satisfactorily elicit the sounds sought for, but further gain valuable information by noting the *amount of resistance* which the finger encounters, and likewise the amount and length of the vibrations communicated to the superimposed digit. Percussion is the most difficult to acquire of the arts used in physical diagnosis, and the maxim that "practice makes perfect" is peculiarly applicable here. The stroke should be delivered from the wrist, as in piano-playing; never from the elbow. The hammer fingers should fall perpendicularly and strike the finger which is used as pleximeter at a right angle to its surface, while the latter is applied as firmly and as evenly to the chest wall as possible. For percussing, the middle and third fingers are superior to the index and middle fingers. The nails should be kept short, that they do not produce sounds by impact. In percussing over the thorax the fingers should be applied to the intercostal spaces parallel with the ribs, never across the rib and interspace, since this gives two notes differing in

quality. Over the clavicle, mediate percussion gives the best result and often gives the earliest indication of disease by a variation from the normal note. Owing to varying conditions, such as thickness of the overlying muscular tissues, quantity of fat present, etc., the sounds elicited by percussion over the lungs vary greatly not only in different areas upon the individual, but likewise over corresponding areas in different individuals; so much so that we are not far wrong in saying that every lung has its own normal note.

PERCUSSION SCALE.

In spite of these variations, we may construct a rough scale which is generally applicable, although the notes lack the fixedness of musical tones. The note of highest pitch is obtained by direct percussion over a bone, as the sternum or clavicle, and is called *osteal* pitch.

Fig. 25—Pleximeter.

Next is the tone produced by percussion over the trachea during quiet inspiration. This is called *tubular* pitch. The note produced by percussion over the second anterior interspace or the axilary region of a healthy, well-developed individual falls next in the scale, and this note is called the *vesicular* note or the *normal pulmonary* note. Flint's comparison of this note to that produced by tapping a large loaf of bread is classic. The note produced by percussion over an air-containing cavity of considerable size, as the stomach and intestine, is called *tympanitic*. It is lower in pitch than the vesicular note. It is not difficult to divide tympanitic resonance into two tones, *small tympany*, the note of the small intestine furnishing the standard therefore, and *large tympany*, the latter being the note obtained by percussion over the empty stomach or the colon. The note which Flint called *vesiculo-tympanitic* occurs in disease, but in the scale of pitch assumes a place between the normal vesicular resonance and small tympany.

Two other sounds remain to be considered, *dullness* and *flatness*. Gee defines dullness as the absence of tone, tone being that succession of sound impulses which characterizes a musical tone, but which is

absent in a mere noise. Da Costa* defines a dull sound as one denoting the absence of air. "It is the sound of both fluids and solids. It is thus the sound sent forth by the airless viscera; from the liver, spleen and heart." We believe, however, that a profitable distinction can be made between dullness and flatness by calling the sound possessing the above qualities flat, and calling a sound which approaches flatness

Fig. 26—Percussion of dorsal regions. Patient astride of chair with arms folded across the back. Intercostal spaces widened, scapulæ rotated outward.

in pitch, but which still retains some resonance, dull. In the scale of pitch, the terms would fall above osteo-pitch, flatness being the highest possible in the scale. Writers who desire to avoid this distinction use instead of dullness the term impaired resonance, modified by the adjectives "slight," "considerable," and "marked". Their inconsistence is made apparent by the terms "relative dullness," "dullness

*6th Edition, *Medical Diagnosis*, p. 240.

more marked", etc., which are used by most systemic writers on practice.

A protracted discussion of the theories of percussion sounds would be profitless. Two only have received extensive recognition, that of Gee, who believes that the vibrations originate in the bronchi, and that of Bristow, who advocates that the tones are produced solely by vibrations of the chest walls, hence, any interference with these vibrations changes the note.

Scale of Pitch. Arranging the notes on a descending scale, beginning with the apex note, or note of theoretically highest pitch, we have:

Flatness
Dullness
Osteal
Tubular
Vesicular
Vesiculo-tympanitic
Small tympany
Large tympany

It may assist the student somewhat in his conception if we compare these eight variations with the notes in the musical scale from high C to low C, with this reservation, that the resemblance ends with the fact that each of the percussion notes is lower than its predecessor.

While the above subdivision of the percussion notes might seem at first glance to be an unnecessary refinement, the terms are all met with in the works of various standard writers, hence a clear classification of them for the benefit of the student cannot be amiss.

It is to be prefaced that the percussion sounds produced over the pulmonary areas can vary in health only in regard to pitch, intensity and duration. Pitch changes with the number of vibrations per second, the greater the number of vibrations the higher the pitch; while the intensity varies with the amplitude of the vibrations, the law being, the greater the amplitude the louder the sound. Duration varies inversely with pitch, the higher the pitch the shorter the duration of the sound. The pitch also varies with the degree of tension, both of the covering of the lungs and of the organ itself. The rule above given applying again, viz., the greater the tension the higher the pitch, just as in musical instruments, the tighter the string is

tuned up the higher the note. The pitch of the pulmonary percussion sounds is altered by disease either in the direction of dullness or of tympany. Changes in the sounds are best appreciated by careful comparison between corresponding areas of the two lung surfaces, ignoring for the moment the very slight differences which normally exist.

CHANGES IN THE PULMONARY PERCUSSION NOTE PRODUCED BY MORBID PROCESSES.

Increased resonance is frequently met with. It is called hyperresonance. Its quality is that of the normal percussion note more or less exaggerated, that is to say, the *volume* of sound, the intensity and duration are increased, while its pitch is lowered. It means increased volume in the air vesicles without increase in the tension. Temporary over-distension produces this note in its slightest degree, while emphysema produces a more marked example.

Skoda's resonance is a note of higher pitch than true tympany, yet with a tympanitic quality. It occurs in connection with two conditions, pleuritic effusion and pneumonia of the lower lobe. It is most strikingly manifest in the infra-clavicular region in a case of moderate effusion. Osler says, "In the subclavicular region the attention often is aroused at once by a tympanitic note, the so-called Skoda's resonance, which is heard perhaps more commonly in this situation with pleural effusion than in any other condition. It shades insensibly into a flat note in the lower mammary and axillary regions. Skoda's resonance may be obtained also behind, just above the limit of effusion." Tyson maintains that Skoda's resonance and Flint's vesiculotympanitic note are identical.

Tympanitic Resonance. Lower in pitch than the preceding and produced by percussion over a quantity of air, "enclosed in walls which are yielding, but neither tense nor very thick." (Da Costa.) The sound is somewhat musical, and the pitch varies with the size of the cavity. It is abnormally present in pneumo-thorax, or over large pulmonary cavities.

Diminished Resonance. Impaired resonance, which in our scale we have denominated dullness, is a note of higher pitch than the normal vesicular sound, but the *duration* of the note is shorter and sharper. The note closely resembles that produced by percussion over a thin board; hence its quality is described as wooden. It is noticeable in partially consolidated lung, in compression or displacement by

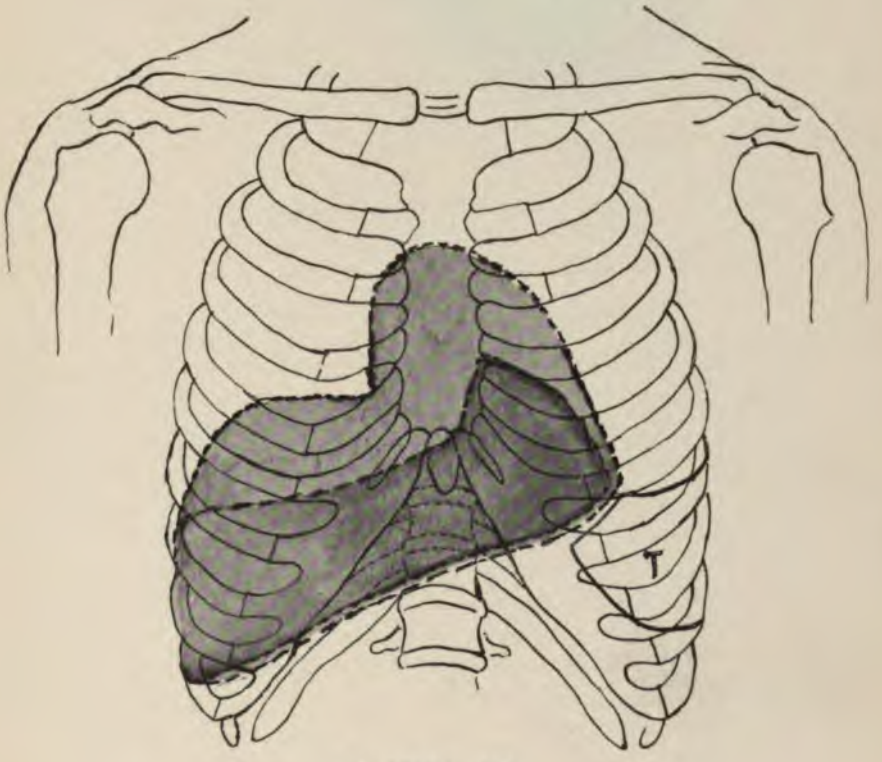


PLATE III.

RELATIVE AND ABSOLUTE PERCUSSION AREAS.

The shaded area represents the area of flatness of heart and liver, fusing into one.

The dotted area marks out the area of heart and liver dullness obtained by deep percussion.

The lower dotted line represents the descent of the liver with forced inspiration.

T. Traube's tympanitic or semi-lunar space.

morbid growths. Increased resistance generally accompanies it. Even over wholly consolidated lung the note is not entirely flat so long as any vesicular substance remains.

Flatness is the absence of tone, absence of resonance, and is the height of pitch. Such a sound is heard when percussion is made over the thigh or over a large pleuritic effusion.

Amphoric resonance is metallic in quality, while tympanitic in pitch. It indicates a large hollow space with firm elastic walls, as a cavity which communicates with the bronchus. Percussion over the trachea with the patient's mouth partly open furnishes a fair imitation of the amphoric quality.

The Cracked-Pot Sound. Bruit-de-pot-felé of the French writers. Its quality is indicated by the name. This sound is closely associated with amphoric resonance. The condition usually occasioning this sound is a cavity freely communicating with the bronchus, the cavity having yielding walls. To produce the sound it is necessary to strike a strong, sharp percussion blow while the patient holds the mouth open. It may be imitated by striking the loosely folded hands across the knee. Da Costa says the sound is met with independent of excavations in "pleurisy above the seat of the effusion; in bronchitis of children; near a pericardial exudation; in emphysema and certain instances of pneumothorax. Indeed, any disorder in which the chest wall remains very yielding, and in which a certain amount of air contained in the lung or pleura and in uninterrupted connection with the external air is, by sudden percussion, forced into a bronchial tube, will occasion this cracked-metal sound."

We briefly indicate again the outlines of the lungs, (see also section on Divisions of the Chest). On the back the lungs extend from the seventh cervical vertebra (vertebra prominens) to the level of the bottom of the tenth dorsal. In front the apices extend for varying distances above the clavicle, from a half inch to one and a half inches in extreme cases. On the right side we encounter hepatic dullness about one and a half inches below the nipple. On the left side cardiac dullness begins at the lower border of the third rib. In the left lateral region we encounter stomach tympany and a little in front of the mid-axillary line splenic dullness begins at the ninth rib. Anteriorly, along the right side of the sternum, the right lung extends as low as the sixth costo-sternal cartilage. The left overlaps the heart as far as the fourth cartilage. In the mid-axillary line the edge of the

lung reaches the lower border of the seventh rib. The liver dullness is therefore indicated by a line beginning at the junction of the fifth cartilage with the right side of the sternum, and passing over the seventh rib in mid-axilla, the lower body of the tenth posteriorly to the eleventh vertebra. It should be remembered that in the erect position the spine of the tenth vertebra is on a level with the middle of the body of the eleventh. The bifurcation of the trachea is behind the sternum, opposite the second costosternal cartilage and the arch of the aorta curves from right to left and from before backwards over this division.

AUSCULTATION.

Auscultation is the recognition by the ear and the determination of the significance of sounds emanating from normal or diseased structures. It is, therefore, almost necessarily limited to the respiratory and circulatory systems. The late Professor Flint picturesquely described it as a kind of eaves-dropping.

Auscultation may be mediate, by means of the stethoscope, or immediate, by the direct application of the ear. In practice, auscultation should follow percussion. In using the instrument care should be taken to avoid pressing the bell too firmly against the tissues as false sounds, resembling friction sounds, thus may be easily produced. In the case of children the premature exhibition of the stethoscope may cause fright, and, as sounds of all kinds are readily transmitted through the thin chest walls of the young, often its use may be dispensed with. In the examination of the lungs the stethoscope is sometimes of doubtful advantage as compared with immediate auscultation, and as the practitioner frequently may be caught minus this portion of his armamentarium, it is wise to practise both forms of auscultation. One sometimes encounters cases, especially in hospital and dispensary routine, in which he is loath to apply the ear, even with towel or napkin intervening, and occasionally over-modest female clients make objection to the procedure. It is to be observed that in the case of females the configuration of the breasts renders immediate auscultation more difficult and less accurate than mediate; likewise in the examination of the heart in both males and females, by reason of the circumscribed areas of the valvular sounds, the stethoscope is almost a necessity not only for their accurate location and differentiation but also that sounds emanating from neighboring organs, as

the lungs and pleura, may be excluded more readily. If it is desirable to cover the chest during the examination, see that the covering be of such nature as not to interfere with the transmission of sound nor of a material of itself to originate sounds. The best covering for the purpose is a thin, gauze undervest; in the absence of which, a thin, soft towel is a fair substitute. Starched garments are especially to be avoided. The author has known the soft creaking of a starched chemise, freshly donned in anticipation of the examination, to be mistaken for the friction sound of beginning pleurisy.

The most reliable evidence is gathered by repeated examnations of corresponding areas, causing the patient alternately to breathe naturally, deeply, to cough and to count, as occasion requires. I have found that repeating the number "nine hundred and ninety nine," by reason of its ringing character, to be especially suitable in testing vocal and tactile fremitus.

NORMAL AND ALTERED RESPIRATORY SOUNDS.

It is manifestly impossible to recognize alterations in the pulmonic or cardiac sounds unless we are thoroughly familiar with the normal ones. Constant study of healthy respiratory and heart sounds is the only way in which the ear can be kept in accord with the demands made upon it. Just as the musician attunes his instrument before playing, so the diagnostician should constantly attune his ear.

The varieties of respiratory sounds which are recognized in health are of three types, but the unfortunate lack of uniformity in nomenclature is not only most confusing, but would lead the student to conclude that there are half a dozen varieties.

It must be emphasized that the sounds about to be described are natural, and only have a pathologic significance when heard outside of their own proper spheres, or when one which should be heard in a certain locality is replaced by another.

As a starting point, we begin with the air sounds heard over the glottis or larynx. Here the ear recognizes a harsh, blowing sound of high pitch and great intensity, heard both on inspiration and expiration, which acts are separated from each other by a distinct silent interval. The quality of this sound is unmistakably blowing, tubular, and somewhat hollow. As we descend from the larynx to the top of the sternum, the sound undergoes a very slight modification. This sound is known as *tracheal, bronchial,* or *tubal or breathing*: the vari-

ous terms being used by different authors as having the same significance and being interchangeable.

If next we apply the instrument over the second interspace, anteriorly, we hear a much softer sound. Its pitch is decidedly lower, the blowing character has been replaced by a gentle breezy rustling of short duration, heard best with inspiration, followed *without any appreciable interval* by a still softer *expiratory* murmur of yet lower pitch, shorter duration, but with a faint suggestion of blowing. As this murmur is supposed to originate in the alveoli, it is called *vesicular breathing*. It is best heard as well as most characteristic at the point indicated. At the apices and low down posteriorly its intensity diminishes, but its quality remains unaltered. This is what is meant by the normal breath sound, or the normal respiratory murmur, and the student will do well to fix indelibly in his mind its qualities.

The third recognizable type falls between the two just described, and shares the qualities of each. It is a sound heard over the subdivisions of the trachea, that is to say the roots of the lungs: hence is heard normally over the manubrium in front and in the interscapular region behind as far down as the top of the fifth dorsal vertebra. With inspiration the vesicular element is diminished but not absent. Inspiration is separated from expiration by a distinct interval, shorter than in the first variety. Pitch and intensity likewise fall between the two varieties above described. This form of respiration is known as *broncho-vesicular* breathing.

CHANGES IN THE BREATHING SOUNDS PRODUCED BY DISEASE.

I. The vesicular murmur may be altered in intensity, by being increased or diminished, or the murmur may be suppressed.

Increase in the intensity of the vesicular murmur is called *puerile breathing*, on account of its likeness to the breath sounds normally heard in children. It denotes increased activity and follows violent exercise. It is not necessarily a sign of disease. Inspiration and expiration are equally affected. When heard in adults it indicates that an increase of function in the unaffected portion of the lungs is compensating for diminished activity elsewhere. It is often called *supplemental* breathing or *exaggerated* breathing. One of the best examples of its occurrence in disease is the respiratory murmur heard over the sound side in cases of pleuritic effusion.

II. *Diminished Breathing.* Diminished or feeble respiration is the opposite condition to puerile breathing. It is due to diminished functional activity, caused by obstruction to the entrance of air into the vesicles; by partial obliteration of the cells by deposits within, or from pressure upon them. The character of the normal breath sounds is unaltered, but the intensity is diminished. It may arise in the course of a variety of affections, as paralysis or pleurodynia. Feeble respiration at the apex combined with impaired percussion resonance is a strong indication of the early stage of tuberculosis.

III. *Absence of the Respiratory Murmur.* Large pleural effusions, massive pneumonia in which the secretion totally occludes the bronchi, and collapse of the lung, are the only conditions likely to cause entire absence of the breath sounds.

IV. The respiratory rhythm may be so changed that the relative length of inspiration to expiration is altered; or the respirations may become uneven and jerky, exhibiting the so-called wave or cog-wheel character. In the first instance it is the expiration which is most affected and the change is usually in the nature of a prolongation of that act. The prolongation is generally accompanied by a change of pitch. If the pitch be elevated it means consolidation, deposits within the air cells or in the nearby bronchi. Here the quality of the sound gradually ascends to bronchial or tubular. If the pitch is low and the quality unchanged, it indicates emphysema. In the one case the bronchial deposits mechanically dam back the air; in the other the loss of elasticity causes the expiratory prolongation.

Cogwheel Respiration. The inspiration is disconnected, broken into two or three parts due to the uneven expansion. Affections of the muscles of respiration or previous disease of the lungs or pleura, especially old adhesions, or diseases of the bronchi offering an impediment to the entrance of air are the causes. It has been met with in hysteria, in intercostal neuralgia and other spasmodic affections, as well as in pulmonary tubercular deposits of which it was once considered a pathognomonic sign. Here its favorite seat is near the apices.

Bronchial Breathing. The character of this respiration has been already described. When heard over the vesicular areas of the lungs as the result of disease the qualities above attributed to it are little altered but the *intensity* is diminished. The tracheal sound as heard at the root of the neck posteriorly is the prototype of bronchial respira-

tion and should be used as the standard of comparison. Bronchial breathing is higher in pitch than the vesicular inspiratory sound and retains more of its tracheal qualities. Its expiratory quality is affected in the same way, its intensity increased, its duration equal to or longer than inspiration, its pitch elevated, and inspiration and expiration separated by a distinct interval. Bronchial breathing signifies the consolidation of the vesicular structure from deposits within, or air-cell obliteration from pressure from without. The sounds of the still patent, large-caliber bronchi are then conducted through the denser tissue to the ear, replacing the vesicular murmur and causing it to appear as though the sounds originated immediately under the ear.

Sometimes adventitious growths, as solid tumors or aneurysms, intervening between the chest wall and the trachea or a large bronchus, will cause obliteration of the air cells and the bronchial bruit is then heard over the tumor area.

As indicating consolidation, bronchial breathing is heard in pneumonia and in the infiltrated areas of phthisis. As the result of intrathoracic pressure it is heard over the compressed lung above moderate and large effusions.

Broncho-vesicular Breathing. When heard peripherally in diseased conditions it portends partial consolidation, but less in degree than that indicated by bronchial respiration. It is subject to variations in quality, especially intensity, and may pass into the bronchial type by exacerbation of the conditions which cause it. As the change occurs, more and more of the vesicular element is lost and the tracheal quality assumed, as indicated by an ascent of both intensity and pitch.

Tubular Breathing, as the term is generally used, is synonymous with bronchial breathing. It indicates the breathing of complete consolidation, as does the latter, but the former term is more limited in its application. Its quality is described as whiffling, and its pitch as higher than the bronchial pitch. Like the latter tubular breathing is present in pneumonic consolidation and in the complete consolidations sometimes met with in the superficial areas of phthisis.

Cavernous Breathing, as its name indicates, is produced by the entrance and exit of air from a cavity. The cavity may be within the lung substance or external to it, without causing alteration of the cavernous sound, provided it be entirely or partially empty and that it

communicates with an open bronchus. It is asserted that a cavity must be as large as a walnut to produce the phenomena attributed to it.

The inspiration is low-pitched, *hollow* and *blowing*, as is the expiratory sound. Often, but not invariably, expiration is still lower in pitch than inspiration, which is the reverse of bronchial breathing. The pitch is never raised on expiration and expiration is longer than inspiration. It requires for its production yielding walls. As a standard of comparison for cavernous breathing, we compare it with the sound heard at the lower end of the trachea. The sound is often associated with gurgling, due to fluids confined in the vomicæ, which may disappear when the cavity is entirely filled, to reappear after expectoration. It is significant of the third stage of phthisis, of bronchiectasis or of pulmonary abscess.

Amphoric Breathing, like cavernous, requires a cavity with resilient walls communicating with a bronchus. It is a high-pitched, blowing sound, the qualities being those of cavernous breathing in an exaggerated degree. But in addition thereto it has an echoing, metallic character, as has the corresponding percussion note. It may be imitated by blowing across the mouth of a bottle or jug (Amphora). Its intensity varies. It indicates a large cavity and usually means pneumothorax. Rarely a pulmonary cavity may afford the necessary conditions for its production.

NEW OR ADVENTITIOUS SOUNDS.

In addition to the changes and modifications of the respiratory rhythm already described, auscultation reveals certain sounds which are in no wise related to or analogous to the normal sounds. Such are râles, rhonchi, friction sounds, echoes and the like.

Râles are peculiar sounds generated in the air tubes by the passage of air through exudates contained therein, or by the forcible separation of agglutinated surfaces, as the swollen lining of the small bronchi or the collapsed alveoli. When they originate in a portion of the lung which has undergone consolidation, or when due to the formation of a thick, viscid and not easily displaced exudate into the cells, the character of the sound is sharp, crackling, dry, or even explosive. These sounds are described as crepitant or crackling râles, and are gauged according to size and intensity into small, medium and large crepitant râles. As already stated, they mean consolidation of the vesicular portions of the lungs. The small, sharp crackle, heard often-

est with inspiration, originates in the alveoli, and is heard in the early stages of tubercular formation, or may be accepted as the earliest sign of the softening stage of the pneumonic process. This râle is not limited to inspiration, but may also be heard during expiration. The larger crackling râles are in character the same as the small râles and indicate an extension of the associated processes. Hence they are heard in the softening stage of pneumonia and of tubercular deposits. They also occur in broncho-pneumonia. The largest crepitant râles are confined almost entirely to minute cavities, surrounded by areas of consolidation which form during tubercular softening.

Sounds produced in the manner just described, particularly when they emanate from lungs whose structure is little altered, and in which the lesions are confined principally to the lining membrane of the tubes, vary in character with the amount and nature of the secretion. They may originate in any portion of the bronchial tract, including the trachea, but usually are confined to the lesser bronchi. When these sounds are dry and have somewhat of a musical character, they are termed *rhonchi*. When their nature indicates the presence of liquid in the tubes, they are denominated moist, liquid, or mucous râles. Sometimes the sounds suggest the breaking of small bubbles. Such sounds originate in the smallest bronchioles and are heard in capillary bronchitis. Others are larger and have a gurgling character.

All of these sounds are extremely evanescent, changing their location, appearing or disappearing with forced respiration, coughing, or change in the position of the patient. They may be so turbulent as to entirely obscure the normal respiratory murmur. These râles are named small, medium and large bubbling or mucous râles, and the names are sufficiently descriptive of their location and character. The large râles occur in cases involving the trachea or largest bronchi, and are common in chronic conditions, such as tracheitis and bronchitis. They are the cause of the "death rattle" sometimes heard in expiring persons. These râles are heard in all pulmonary diseases in which inflammation of the bronchi is the essential element of the disease or occurs as a complication. Pure, large gurgling râles, having the character of liquid poured from a bottle, are confined to partially filled cavities, and are heard on coughing.

Rhonchi are dry sounds, and like wet râles are confined to the bronchi. They vary in both character and pitch according to their location and the amount of obstruction or narrowing produced by the

causative condition. The narrower the tube, the higher the pitch of the sound. Such narrowing may result from swelling of the membrane, as is apt to be the case in inflammations of the finer divisions, or from secretions, which is the usual cause of their occurrence in the large tubes; or the two causes may operate together, as in bronchitic asthma. The low-pitched rhonchi are called *sonorous*, owing to their reverberating character; the high-pitched are called *sibilant* by reason of their hissing nature. Others have a whistling sound.

They may be heard on either inspiration or expiration, or both, and accompany acute and chronic inflammations of the bronchi. The finer, higher-pitched rhonchi are heard in acute conditions and when exacerbations or extensions of chronic forms take place. They are very variable and large rhonchi may suddenly disappear. The finer varieties, indicating obstruction, are somewhat more stable. As has been said under Cough, they often indicate the first stage of a condition in which moist râles are the second stage. Bronchitis and asthma and emphysema give us the best examples.

Stridor is the name given to the harsh, rough, vibrating sound imparted to the breathing by certain conditions. It might be called respiratory tremor. The breathing is noisy and labored, inspiration is prolonged and the voice is often raucous. Direct pressure on the larynx, trachea or a large bronchus produces it, hence the most frequent cause is thoracic aneurysm or a mediastinal growth. Next in frequency is syphilitic stenosis. If due to local conditions as interlaryngeal growth or paralysis of the vocal cords, an examination of the larynx will reveal the cause.

Friction Sounds. These sounds result from the rubbing together of two inflamed pleural or pericardial surfaces. In older cases the surfaces are roughened from the deposit of exudate, or fine fibrous union may have formed.

Friction sounds consist of a number of short, repeated sounds of a crackling, rubbing or crepitant nature. They are often compared with the creaking of new leather. They are usually heard over a very limited area, possibly never exceeding in size a half dollar. They are unilateral and oftenest confined to the lower thoracic areas.

Pleural friction sounds are generally heard on both inspiration and expiration, but may appear only at the end of forced inspiration. The sounds are frequently referred to as "come and go" sounds. It is

often difficult to decide whether the sound arises from alteration of the pericardial sac or of the pleural membrane.

The following points will aid in the distinction:—

The location; the evident superficial situation of the pleural sounds.

The relation of the two sounds to respiration above mentioned: pericardial sounds do not disappear when respiration is suspended.

From the crepitant râle it is distinguished by the fact that pleural sounds are influenced by neither cough nor by respiration.

That deep respiration increases the pain in the case of friction.

That friction areas are more circumscribed and that the râle is heard only on inspiration.

AUSCULTATION OF THE VOICE SOUNDS.

When the ear is applied to the normal chest while the patient speaks a distant, muffled, humming, vibratory sound is heard. The spoken words are inarticulate. The sound is strongest in deep-chested adult males and weakens by gradations in women and children. Whatever the character of the sound may be, it is pretty constant in pitch and intensity over the entire pulmonary periphery of the individual. The sound is called *normal vocal resonance*. It is more intense in the vicinity of the trachea and large bronchi and is slightly more intense in the right infraclavicular region than in the corresponding region on the left. Conditions which increase conductivity increase the intensity of the sound and *vice versa*. Hence consolidation of the lung of any degree whatsoever increases it.

The pitch is unaltered in this case but the sounds are louder. On the other hand emphysema, in which the lung is, so to speak, *rarefied*, diminishes the vocal resonance, as does also pneumothorax, by separating the conducting substance from the chest wall. Thickened pleura similarly affects it by offering a barrier, and over pleural effusions it is totally absent.

Occlusion of the bronchi by mediastinal growths, aneurysm or other causes, prevents the sound waves from reaching the periphery, hence vocal resonance is also absent in these conditions.

This absence will be understood readily when it is considered that the vibrations set up by the act of speaking must reach the surface by traversing the intervening air column. If the column is interrupted by bronchial obliteration, the sound waves will be absent over the lung

surface so supplied and likewise over the corresponding area of lung covering.

Bronchophony. Over the large bronchi the voice sounds heard in health are of greater intensity, more sonorous, more concentrated and of higher pitch than those sounds heard over the vesicular regions. This is called the bronchial voice and is the type of bronchophony. The sound seems to be near the ear. The term bronchophony means, therefore, increased or augmented vocal resonance. It bears the same relation to normal vocal resonance as does bronchial breathing to the normal vesicular breath sounds, and when heard in localities other than over the roots of the lungs means increased conductivity, hence consolidation. The sounds are inarticulate.

Pectoriloquy is the name given by Laennec to the distinct transmission of articulate words through the chest to the ear. By some it is regarded simply as exaggerated bronchophony. In character the sound closely resembles the sound of the voice heard over the larynx. In bronchophony, as stated, the sounds are inarticulate, while in pectoriloquy not only is sound of like quality transmitted to the ear, but the articulate words are audible. In a few cases the sounds not only seem to arise under the ear, but are much intensified and the pitch elevated. This is a union of bronchophony and pectoriloquy and is named bronchophonic pectoriloquy.

In other instances the character of the voice sounds, although articulate, are distant, hollow and even may be slightly ringing. Here the pitch is not elevated, the area is limited and the words do not seem to arise under the stethoscope. This Flint named cavernous pectoriloquy. He says that the former means solidification, the latter a cavity.

Amphoric Voice is so named when in addition to being ringing and hollow it has also a musical, a metallic or a tinkling character. The sounds are not articulate as in pectoriloquy. It is generally taken to mean pneumo-hydrothorax, but may occur in large cavities offering similar conditions, especially large vomicæ whose walls are formed by limiting membrane.

Egophony is bleating bronchophony, caused by a thin layer of fluid set into vibration by the voice. It is of very rare occurrence, but has been found in cases of moderate pleural effusion, its usual seat being at the lower angle of the scapula.

The Whispered Voice changes or is modified by disease as well as

the spoken voice. Normally the whisper resonance is not heard except over the large bronchi which form the roots of the lungs, and the adjacent areas in the upper thorax. Here is it a soft, blowing sound which accompanies each word. Increased or *exaggerated whispered resonance* and *whispering bronchophony* may be said to be present when the normal whisper just described is heard over the vesicular areas of the chest, where they are never normally present. The whispered sounds then have the characteristics of their spoken congeners. The second of these two is higher in pitch, more intense and nearer the ear than the first. It indicates consolidation.

Also may we have whispering pectoriloquy, amphoric whisper, and cavernous whisper. Whispering pectoriloquy is a surer indication of cavity than its spoken equivalent. The whispered voice is sometimes transmitted in pleurisy when the spoken voice is not. Bacelli considers this sign as diagnostic between serous and purulent effusions, but clinical experience does not bear him out. It is however perpetuated as Bacelli's sign.

There still remain to be considered a few adventitious sounds of somewhat rarer occurrence than those already described.

Crepitation. A fine, crackling sound exactly imitated by rubbing the hair between the fingers, close to the ear. It has long been accepted as the earliest sign of fibrinous deposit within the air cells in pneumonia. It may also occur when air enters the collapsed air vesicles and is due to the separation of their walls, hence is heard at the end of inspiration (see Pneumonia). It may be present also in hypostatic pneumonia, œdema of the lungs or in areas of lobular collapse.

Succussion Splash. This was described by Hippocrates, and is a peculiar splashing sound produced by violent coughing or a sudden shaking of the body. A large cavity containing both air and fluid is necessary for its production. It may be exactly imitated by splashing an uncorked earthenware jug containing a small quantity of water. It is present in hydro- or pyo-pneumothorax (q. v.).

Coin Ring or *Bell Sound* may be produced in pneumothorax or in very large pulmonary cavities. A coin is placed flat against the chest and is struck with the edge of another coin; with the stethoscope applied over the affected area, is heard a clear, bell-like tinkle.

Metallic Tinkling is a sound which Loomis likened to dropping pins or small shot into a metallic vase. The sound is echo-like, clear, high-pitched and ringing. It may be single or a series of sounds, may

be produced by the movements of respiration, by coughing or even by speaking. A large, dense-walled pulmonary or pleural cavity and a communicating bronchus are necessary for its production. It is oftenest heard in pneumo-hydrothorax. Dr. Walsh regards it as due to the echo of bursting bubbles shut up in the cavity. It is also attributed to dripping fluid striking upon a liquid surface. The term am-

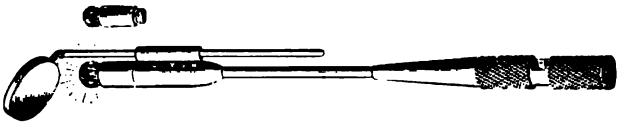


Fig. 27.—Laryngeal mirror with electrical connections.

phoric echo is used in the same connection to describe the above sound, or one very similar.

Post-tussive Suction, a rare but valuable sign of cavity. When an excavation is so conditioned as to be compressed by the act of coughing, the air is expelled therefrom during that act with a slight hissing noise. The dilatation of the cavity after compression is sometimes followed by an audible, air-suction sound, varying in intensity with the size of the cavity. The name is sufficiently descriptive of its quality. A sound similar to the above, also produced by air entering a cavity

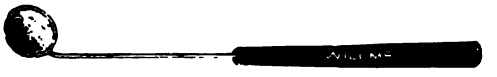


Fig. 28—Laryngeal Mirror.

with inspiration, has been described by Laennec, Skoda and others, and called "the puff".

THE LARYNGOSCOPE.

This well-known instrument enables us to view the interior of the larynx, the vocal cords and a portion of the trachea.

Acute and chronic laryngitis cause hyperæmia, erosions of the cords and occasionally a slight exudate. In the chronic form the membrane is thickened. Œdema and pseudo-membranous formations give names to their respective varieties.

Tuberculous Inflammation. The vocal cords are [illegible] and eroded, the movements restricted; infiltration, superficial and deep, is

seen. Thickening of the arytenoids is the earliest sign. Other symptoms of tuberculosis coexist.

Laryngeal Syphilis. Symmetrical superficial ulcers occur early. Gumma, deep ulceration and necrosis of the cartilages are tertiary.

Paralysis of the laryngeal muscles arises from various central lesions, medullary syphilis, multiple sclerosis, locomotor ataxia and hysteria. In another group of paralyses the lesion affects the fibres of the recurrent laryngeal in their tortuous course, or the laryngeal fibres in the vagus or accessory nerve, or the direct laryngeal nerve is subject to pressure. New-growths are the principal cause of such paralysis.

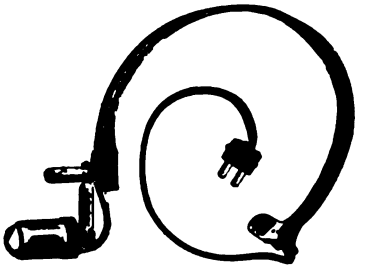


Fig. 29—Laryngeal Headlight.

The left nerve is most exposed to injury owing to its course around the aorta. Mention is made of the condition under Aneurysm.

Causes. Aneurysm, pleural thickening, mediastinal tumors, enlarged bronchial glands, carcinoma of the œsophagus, enlarged thyroid and even pericardial effusions are assigned as peripheral causes of the lesion.

THE OPHTHALMOSCOPE.

The examination of the eye-ground by means of this instrument adds greatly to our diagnostic knowledge. Much information is within reach of the general observer even though he has not received special training in technic.

Besides diseases strictly pertaining to the eye, the following conditions are worthy of attention.

Choroiditis. Syphilis, rheumatism and gout often originate the disease. Typhoid and puerperal fevers, septicæmia and conditions causing thrombosis are sometimes complicated by it.

Tubercle of the choroid is observable in miliary tuberculosis;

gumma in syphilis. Sarcoma of the choroid is met with occasionally, the subjects being usually above thirty-five years of age.

Pulsation of the retinal veins occurs during cardiac diastole but is physiologic. Pulsation of the arteries is generally pathologic. It is seen in states of increased or diminished arterial tension and occurs in aortic regurgitation with hypertrophy, in Basedow's disease, in syncope after hæmorrhage.

Papillitis is most frequently caused by intracranial diseases, especially cerebral tumors or meningitis, but glycosuria, albuminuria,

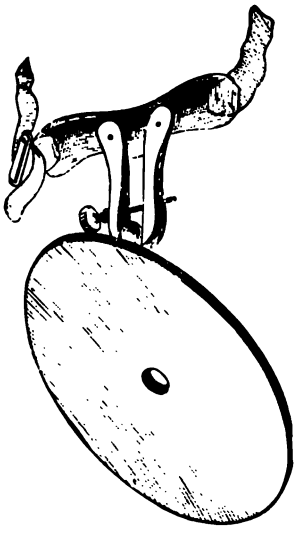


Fig. 30—Laryngeal Reflector.

lead-poisoning, anæmia, amenorrhœa and syphilis also are recognized causes.

Atrophy of the optic nerve occurs in spinal diseases as tabes dorsalis, in syphilis, diabetes and malaria. Toxic agents, alcohol, tobacco and lead are designated as causative.

Cardiac valvular diseases may cause embolism of the retinal artery, as may also albuminuria and pregnancy.

Retinal hæmorrhage may result from numerous derangements of the vascular system, as arterial sclerosis, valvular diseases, especially mitral disease, embolism and thrombosis, miliary aneurysm and the

following general conditions: Diabetes, albuminuria, pernicious anæmia, purpura, scurvy and leukæmia.

Retinitis is caused by albuminuria, glycosuria or syphilis. The

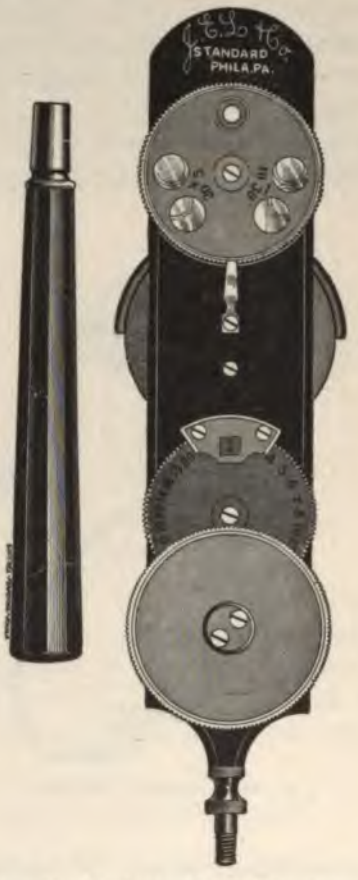


Fig. 31—Morton's Ophthalmoscope.

white patches upon the retina—albuminuric retinitis—which are seen in Bright's disease have often revealed the malady when entirely unsuspected.

Cataract. Diabetes is a well established cause of cataract.

SECTION IV.

SYMPTOMS OF PATHOLOGIC CONDITIONS OF THE CHEST.

COUGH.

Among the adventitious sounds one which plays a most prominent rôle is cough, and while its presence does not always indicate derangements of the respiratory apparatus yet by reason of its paramount importance as a physical sign, and its almost universal association with all pathologic changes, however slight, in the breathing and vocal apparatus, it is best considered along with the pulmonary adventitious sounds.

Cough is a sudden, single or multiple violent expiratory effort, spasmodic in origin, with the object of expelling some irritating substance from the air passages. Each cough is accompanied by a sudden opening and closure of the glottis so that the air is expelled in forcible blasts.

The character of a cough varies greatly, not only during the course of any single disease, but varies likewise with the multifarious pathologic conditions which originate it. In the beginning of some diseases it is so characteristic that the nature of the affection may be foretold by the cough alone. With the progress of the disease this distinctiveness is often lost, as in phthisis, while in other maladies, as whooping cough, it develops as the disease progresses.

Coughs are *dry* or *moist* as to whether they are or are not accompanied by expectoration. According to the location of the disease which originates it cough is spoken of as laryngeal, tracheal, bronchial, cardiac or sympathetic. The possibility of stomach cough in the sense used by the older writers is now generally denied.

Dry coughs are indicative of the primary stage of almost all inflammatory affections of the larynx and pulmonary system. In pleurisy and the early stage of phthisis, dry cough is significant.

Coughs due to pressure of various growths upon nerve trunks

associated with the respiratory tract, particularly such as involve the recurrent laryngeal nerve, the cough of cardiac affections, thoracic aneurysms and some other conditions, remains dry throughout its course or is accompanied by so little expectoration as to merit that distinction. Irritation of the air passages, whether inflammatory or non-inflammatory, will likewise cause dry cough, as nasal polypi, rhinitis, acute and chronic inflammation of the fauces or tonsils, elongated uvula and hypertrophied tonsils. A similar cough is caused by many ailments the seat of which is entirely removed from the air tracts, and while the relation of cause and effect is indubitable, yet from the manner of their production they must be classified as sympathetic. Thus, dentition, intestinal parasites, various organic diseases of the brain, the stomach, the intestines, the heart or the blood vessels number cough among their symptoms. Diseases productive of dropsies, such as changes in the peritoneum, kidneys, liver and valvular diseases of the heart likewise manifest this physical sign. It is a common manifestation of hysteria and states of high nervous excitement.

Dry cough is of a peculiar irritant character, affecting alike both possessor and listener. I have sometimes attributed this impression on 'he listener to the undefined sense of wasted effort.

A careful study of the varying character of cough in its many phases is instructive. Laryngeal cough is ringing and brassy, although the voice is husky; nasal and pharyngeal coughs are "hawking and hemming" in character. The cough of croup is so characteristic as to originate the adjective "croupy." That of false croup is husky, raucous, stridulous and brassy. The most noticeable quality of chronic laryngeal cough is hoarseness. Likewise vocal hoarseness accompanies most other laryngeal disorders. The cough of emphysema is loud, harsh, wheezing and paroxysmal. The characteristic spasmodic cough of whooping-cough is so well known as scarcely to need description. It consists of a violent, protracted series of abrupt, forcible expiratory efforts, followed by a long-drawn inspiration which is accompanied by a hoarse noise called the whoop, from the similarity of the sound to the syllable. The cough usually continues with brief interruptions until expectoration or vomiting supervenes, only to be repeated again after varying brief intervals. Prior to the development of the whoop, which appears at the end of the first week, the disease presents no signs which distinguish it from ordinary catarrhal bronchitis, more or less of which is associated with it throughout its course.

Commonly associated with certain coughs is an annoying tickling sensation in the throat, the patient frequently observing that if the tickling were stopped, the cough would disappear.

Cough is affected by position, being usually worse during the recumbent posture; by time, being usually worse at night, or, as in chronic bronchitis and phthisis, worse in the early morning.

EXPECTORATION.

Expectoration is the consummation of such coughs as progress through an orderly sequence or which belong to the series that we have designated as *moist* coughs. The matter expectorated often possesses such physical, chemic or microscopic characteristics as positively to identify the source and character of the disease, as in phthisis, pneumonia, bronchitis, abscess of the lung and a few others.

The amount of sputum expectorated varies largely with the disease and may reach 1000 cc. in twenty-four hours. The consistency of the sputum varies greatly, as a rule it is less when the amount is great. The consistency is extreme in the first stage of acute pneumonia, bronchitis and phthisis. In these cases, especially in pneumonia, it is a familiar fact that the sputum cup may be inverted without loss of the jelly-like contents. Sputum containing air floats, while denser, airless masses sink. Very dense sputum assumes round or flat, disc-like shapes, and when such masses float in thinner expectoration, we have the coin-like or nummular sputum, of which the cavities of tuberculosis give us the best examples. Small, cheesy particles which sink to the bottom of the cup are likewise indicative of phthisis and usually contain large numbers of tubercle bacilli. In cases of pulmonary œdema the fluid expectorated is thin, serum-like and covered with froth. The sputa of pulmonary gangrene, of perforated empyema and of pulmonary abscess may be composed almost entirely of pus, which may possess a strong, distinctive odor. The color of the sputa varies from the transparency of mucoid expectoration to the dark-brown or red of admixed blood. The white color indicates leucocytes, yellow and green sputa are purulent, bile pigment also gives a green color to the expectoration. Red denotes blood. Pulmonary gangrene gives rise to a fetid, chocolate-colored sputum. This form of sputum contains various elements deserving of more particular mention, such as elastic tissue fibers, the presence of which always denotes some destructive process in the pulmonary tissue. Such destruction occurs in

phthisis and abscess as well as in gangrene. In former days the recognition of these fibers was, taken in connection with other physical signs, the surest indication of phthisis at the command of the diagnostician. Fibrinous casts are found in the sputa in the course of pneumonias and sometimes in bronchitis. They may be recognized by the eye and beautifully demonstrated by shaking them with a little water in a large test tube.

The sputum of bronchial asthma is very distinctive and finds no counterpart in any other affection. It contains small, translucent pellets or gelatinous masses, named by Laennec, "perles". These ball-like masses, when unfolded, are found to be mucous moulds of the smaller tubes. The entire sputum may consist of these bodies floating in thin mucus. If a portion of the sputum be spread on glass and held over a dark background, it will be found that among these masses are some which have a twisted or spiral shape recognizable by the naked eye, others, when unravelled and viewed under the microscope present the same structure. They assume one of two forms. In the first a few mucin fibrils are twisted upon each other and enclosed in their meshes are entangled a small number of leucocytes, generally eosinophiles of large size and containing numerous fine granules stainable with eosin. In the second form the mucin fibrils are much more numerous and are tightly twisted around a crooked central fiber. Enclosed within the skein are a few cells. Curschmann views the threads as transformed mucin formed in the finest tubes as the result of a bronchiolitis. It has been stated that these bodies have been found in the sputa of chronic bronchitis and of croupous pneumonia, but Osler says he has never found them therein.

The above-described sputa often contain pointed octahedral crystals first described by Leyden and called Charcot-Leyden crystals. They occur later in the attack, while the spirals are found early. If, however, the sputum containing the spirals be kept for several days it is said that the crystals will develop from the spirals. These crystals are found very occasionally in acute and chronic bronchitis and phthisis. Under the microscope they appear either as small, straight hexagonal prisms or as granules of varying size; they are quite generally associated with free eosinophilic leucocytes as already stated. The crystals are found in the blood in myelogenous leukæmia.

The boiled sago-like grains which often appear in the sputum are due to the presence of alveolar cells which have undergone myelin

degeneration and are merely indicative of a catarrhal process without other distinctiveness, although they are often encountered in early phthisis.

The presence of tubercle bacilli and the diplococcus pneumoniæ, and the methods of demonstrating them by stains, are discussed under their appropriate headings.

HÆMOPTYSIS.

Definition. Hæmoptysis is the expectoration of blood which has escaped into the air passages. Occasionaly blood escapes into a cavity which is not in communication with a bronchus, in which case there is no expectoration of blood. Thus there may be extravasation of blood into the pleural sac or a pulmonary aneurysm may rupture into a cavity which has no communication with the air tubes.

Causes. The most common causes of hæmoptysis are:

(a) The rupture of an aneurysm on some branch of the pulmonary artery.

(b) Erosion of a branch of the pulmonary artery due to disease of whatever nature. Such hæmorrhages occur during the advanced stages of phthisis, bronchiectasis, cancer and occasionaly in gangrene of the lung.

(c) Active or passive hyperæmia of either the bronchial or pulmonary capillaries. In this case the quantity of blood is small compared with the other lesions. Such hæmoptysis occurs at the outset of phthisis, in acute broncho-pneumonic phthisis, in acute miliary tuberculosis, bronchiectasis, cirrhosis of the lung, emphysema, in the initial stages of pneumonia and bronchitis; in pulmonary gangrene, cancer, abscess, in short, all inflammatory diseases of the lung. In these cases the quantity is often only sufficient to streak the sputa.

(d) Injuries to the lungs and pleura, tumors and parasitic invasions and all forms of pleurisy may give rise to hæmorrhage.

(e) In young, healthy persons hæmoptysis may arise without any assignable cause, without warning, continue a few days and pass away without leaving any subsequent token of its visitation, and the attack may never be repeated.

(f) Hæmoptysis occurring with lesions of the cardiac valves, particularly in mitral insufficiency and mitral stenosis and in aortic insufficiency. Here it may be profuse and recur at regular intervals for years.

(g) Hæmoptysis accompanying certain impoverishments of the blood, particularly rickets, scurvy, purpura, hæmophilia and occasionally leucocythæmia. Under this head may be included the hæmoptysis of malignant fevers.

Ulcerative affections of the larynx, trachea and bronchi give rise to hæmorrhages which may be profuse and rapidly fatal. Such ulceration may occur even in bronchitis. Aneurysm of the aorta may ulcerate into a bronchus or the trachea. Vicarious hæmoptysis replacing menstruation is too well established to be denied. A recurrent hæmoptysis of arthritic subjects is described by Sir Andrew Clark. In these cases the patient is beyond fifty years of age, the hæmorrhages recur without any serious disease of the lungs being present or developing after the attack.

Character of the expectorated blood. The quantity varies from a mere show to a litre, the rupture of an aortic aneurysm into a bronchus may amount to the latter quantity. The blood is bright-red when moderate in amount, but if enormous may be dark or venous-colored. It is mixed with the bronchial secretions formed in the various diseases with which it occurs. When in any considerable amount it is frothy from admixture of air. The blood of hæmoptysis is alkaline in reaction, a point which serves to distinguish it from blood derived from the stomach. If it is retained for some time within the lungs it coagulates and its color is dark-brown or black. It is then often coughed up in strings or moulds of the bronchioles. For several days after a hæmorrhage the secretions will be stained with brownish streaks.

Symptoms. These vary with the gravity of the attack. When large quantities of blood are suddenly lost the symptoms are more grave than when a like quantity of blood is more slowly lost. In the graver cases blood may pour out of the nose and mouth or it may be coughed up in gulps. Blood which has been swallowed may be afterwards vomited or be passed by the bowels.

The symptoms are great pallor and anxious expression of face, cold, clammy skin, small feeble pulse, faintness and coldness of the extremities. During the attack there is a decided fall of temperature, but subsequently a febrile reaction sets in, the temperature rapidly rising to 102° or 104° F., then falling by gradations for several successive days. This fever will continue, especially in tuberculous cases, for several days to a week, even in patients where there was little ele-

vation prior to the accident. The hæmorrhages of pulmonary tuberculosis may be separated by wide intervals and some tuberculous cases terminate without any distinct hæmoptysis, while other cases are marked by periodical recurrences.

If hæmorrhage comes from a ruptured pulmonary aneurysm the quantity is apt to be large, but if the termination is not fatal, the opening closes and the danger passes. If a vessel erodes the closure is by clot, which may be dislodged, allowing a repetition of the hæmorrhage at intervals of a day or two, as is often observed.

Diagnosis. Inspection of the front of the chest and auscultation with a stethoscope may be made during the continuance or immediately after the hæmorrhage, but no attempt at percussion nor change in the position of the patient is permissible, neither should he be allowed to speak, cough or even breathe deeply. A few moist râles may be heard towards the apex. A previous knowledge of the presence or absence of tuberculous signs is most useful. The heart may be examined early, in order to ascertain whether or not the valves of that organ be diseased.

Difficulty is often encountered in determining from the history of a case whether a previous attack has been one of hæmoptysis or of hæmatemesis. If the patient is seen during the attack the differentiation is easy. In case of a previous seizure, it should be ascertained whether the patient had suffered from cough, expectoration, shortness of breath and other symptoms referable to the lungs, or whether he had previous to the attack dyspepsia, vomiting or other gastric symptoms, or gives a history of hepatic disease, particularly cirrhosis. Perhaps splenic enlargement may be made out. These three organs are the chief malefactors in hæmatemesis, while the lungs, the heart and the vessels assume the same rôles in hæmoptysis. The history may show whether previous or subsequent to the attack the sputum was streaked with blood, and whether faintness preceded it. Sometimes also, in the case of intelligent patients, one can ascertain other valuable facts, as to whether the attack came on during a fit of coughing or of vomiting, and whether or not the blood came all at once or in successive mouthfuls.

The common recital of the patient in the case of hæmoptysis is that he "felt a tickling in the throat," or "had a salty taste in the mouth, followed by the blood." Such a description does not apply to hæmatemesis.

In hæmatemesis the blood is dark in color, acid in reaction; if it has remained long enough in the stomach to be acted on by the gastric juice it resembles coffee-grounds. It may be mixed with food. It is generally clotted. Vomiting is often preceded by a sense of faintness. The passages subsequent to the attack are tarry, but it must not be forgotten that during an attack of hæmoptysis blood is frequently swallowed, to be afterwards vomited or passed from the bowels. For convenience we arrange the above symptoms in parallel columns, as an aid to differentiation.

Hæmoptysis.	*Hæmatemesis.*
1. History and symptoms of pulmonary disease. Cough, moist râles. Signs of cardiac valvular disease.	1. History of disease of stomach, liver or spleen; enlargement. No physical signs of valve disease.
2. Sputa blood-streaked before and after the attack. Attack comes on during coughing. If vomiting, it is subsequent to seizure.	2. Attack comes on during vomiting, often preceded by faintness. Blood comes *en masse*.
3. Blood aërated, alkaline in reaction, frothy, bright-red in color, clots readily; mixed with pulmonary secretions, pus.	3. Blood dark-brown or black in color, acid in reaction, mixed with food, usually in coarse clots or coffee-grounds.
4. Local physical signs.	4. Melæna and nausea subsequent. Local physical signs.

The following signs of all severe internal hæmorrhages are common to both conditions, viz., restlessness, extreme pallor, a quick, feeble pulse, syncope, subnormal temperature, skin clammy or bedewed with cold perspiration, breathing shallow and feeble but hurried, great thirst. In fatal cases consciousness is apt to be retained longer in hæmoptysis than in hæmatemesis, since the stomach loses its power to expel the blood earlier than do the lungs.

In cases not presenting the ordinary physical signs the larynx should be examined for a ruptured vessel or ulceration, as mentioned under causes.

Syphilis of the trachea and bronchi is also an occasional cause of hæmorrhage, likely to be overlooked.

DYSPNŒA.

Difficult breathing accompanies a variety of disorders of very diverse character. Sometimes it is the direct result of gross lesions of the respiratory or circulatory systems, at others it is due to the presence within the organism of toxic materials and occurs wholly apart from discernible lesions. In still other cases subjective causes, as pain or even hysteria, are responsible for its presence. Certain constitutional vices, as rickets, while not wholly accounting for its oncoming, are yet contributory and augment it when present.

Among diseases of which dyspnœa is a well recognized symptom, asthma, of either the bronchial or cardiac type, stands in the front rank. Of the former variety it is the leading symptom. Furthermore it is seen in all inflammations of the respiratory tract from the Schneiderian membrane to the ultimate air cells. It occurs with coryza, with nasal polypi, hypertrophy of the tonsils, retro-pharyngeal, œsophageal, or fauceal abscess, and quinsy. It is characteristic of obstructive inflammations of the larynx, as diphtheria, of new-growths or paralysis of the organ, and of the spasmodic condition known as laryngismus stridulus. In catarrhal inflammations of the larynx it plays a less prominent rôle. The diseases of the trachea, the bronchi and the lungs of which it is a symptom are more particularly described in this work. It may be said to be associated in varying degree with the entire list, as well as with those of the pleura. Admission of air into the pleural sac, as from ulceration, may cause its sudden onset, as does also perforation from without. Cancer of the respiratory tract, and especially cicatrices resulting from previous ulceration, are included in the etiology.

Its intimate association with all forms of heart disease is well known, and here it often forms a safe barometer indicating plainly, by its rise and fall, not only the state and progress of the disease, but likewise the efficacy of the treatment. It accompanies endocardial and pericardial inflammations. Intrathoracic growths are causes, both directly, through the obstruction which they produce, and indirectly, by their influence upon the respiratory nerve mechanism (see Aneurysm). Acute mediastinal lymphadenitis gives rise to paroxysmal dyspnœa; de Mussy holds that the dyspnœa of whooping cough is due to this cause. Thrombosis and embolism of the pulmonary vessels, as well as cerebral embolism, are occasional but grave causes. Any condition which materially alters the intrathoracic pressure, operating

either from within, as emphysema or effusions into the pleural or pericardial sacs, or operating from without, as tumors, hypertrophies and dropsies below the diaphragm, may likewise cause dyspnœa. Kidney diseases, unaccompanied by dropsy, owing to faulty elimination manifest it, and in uræmia it assumes a peculiar type. Its intercurrence during the course of, or during convalescence from scarlet fever should suggest scarlatinal nephritis. It is present in apoplexy, cerebral tumors and other diseases of both brain and cord. Alcohol and toxic substances absorbed into the blood, as well as changes in the blood itself, are known factors, hence its share in the symptom-group of the anæmias. In progressive pernicious anæmia it progresses with the case and is the type of the so-named anæmic dyspnœa. In large goitres and in Ludwig's angina dyspnœa results from tracheal compression.

The dyspnœa of obesity is a matter of daily observation.

Classification. Dyspnœas may be classified as *mechanical, chemical,* and *nervous.* The first includes all those forms which arise from pressure and obstruction, thereby causing a reduction of the air current; from deformities and from lesions which result in hypostasis.

Chemical causes are toxicity, imperfect aëration and the accumulation of CO_2, as occurs in febrile diseases and faulty elimination.

Nervous and reflex causes include spasmodic cases, affections of the respiratory center, peripheral irritations, and palsies.

In many cases two of these factors unite to produce the dyspnœa. According to its relation to respiratory rhythm we classify dyspnœa as inspiratory or expiratory.

Appearances and Physical Signs. The dyspnœa may be plainly manifested by the expression and attitude of the sufferer, the marked evidences of pain, the appearance of labored breathing, the cyanosis or the pallor of the skin, or the noises which accompany the respiratory acts. The respirations may be lessened in number, but are much oftener increased. Normally the number of respirations is 18 to 20 per minute for men, and 20 for women. In children the number varies with age, being 45 per minute at birth, 25 at the fifth year, decreasing to 18 at the fifteenth year. The number of respirations is influenced in health by the same causes which increase or diminish the pulse rate (see Pulse). The relation of the breathing rate to the pulse rate is one to four, or one to four-and-a-half, in health. The number should be reckoned without unduly attracting the attention of the

patient, since this consciousness involuntarily increases the number. Oft times the movement of the bed-clothes suffices for ascertaining the number. If the breath-pulse ratio above given is much exceeded, we may suspect that the disorder has to do with the respiratory function, while if the pulse-breath ratio is altered materially, the probability is that the circulation is at fault. A previous knowledge of the condition of the patient substantially aids in the appreciation of the situation, since in aggravated cases a complete examination may be impossible. Yet even here valuable information is afforded by visible signs, as pulsation, the presence of œdema, anasarca, signs of pressure, of spasm or obstruction, collections of fluids, or even of exudation into the air cells. Perhaps the heart-sounds may be listened to without greatly adding to the discomfort of the patient. Careful attention to the neck, throat and abdomen is recommended. Dilatation of the nares accompanies inspiratory dyspnœa. The accompanying sounds may aid in locating the seat of the disease, as well as intimating its nature. In obstruction of the larynx or trachea, or spasm of the cords, the sounds are loud, rough, vibrating or snoring, and the auxiliary muscles of respiration are brought into play. Sometimes the sounds suggest a valve-like obstruction to inspiration, and the act ends in an abrupt croak or squeak, as in laryngeal diphtheria. Abscess about the fauces or inflammation of the tonsils imparts a peculiar hoarseness and choking, and is accompanied by incessant efforts to expel the viscid secretions. Affections of the glottis, ulcerations and tumors of the larynx, give to respiration a crowing sound or a stridulous character. Bronchial obstruction, when sufficient to cause considerable disability, imparts a whistling or wheezing note to the respiration, which may be heard only on inspiration, or on both inspiration and expiration. Fluids give rise to râles or gurgling sounds. Pneumothorax sometimes is accompanied by a sighing or gasping breath.

Cough occurs in no inconsiderable proportion of dyspnœas, as readily would be concluded from scanning the list of causes. Fits of coughing, as a rule, increase the dyspnœa, or the dyspnœa may occur only during and after coughing, as in pertussis. In almost all consolidations of pulmonary substance the same observation holds good. Expectoration generally brings relief, as in bronchiectasis.

The term dyspnœa is not, however, confined to the phenomena above described, in which the breathing is manifestly labored, and in which auxiliary aid is sought. The term is quite as applicable to those

diseases in which a decided increase in the number of respirations occurs without particular effort as is seen in pneumonia, in which the rate may rise to 60 or even 80 per minute. This form belongs properly to the second division of our classification, viz., *Chemical Form.*

Allied to the latter form is the dyspnœa which occurs upon exertion. The patient may be quite comfortable so long as he remains quiet or recumbent, but the exertion of sitting up brings on an attack. Such dyspnœa occurs frequently in convalescence, but is not confined within its boundaries. In another variety the patient may be free from discomfort so long as he walks on a level or walks at a moderate pace, but should he attempt a slight ascent, or should the necessity to quicken his pace arise, the attack sets in. This may be the first admonition to the patient that his mechanism is deranged. Dyspnœa in young children often shows itself in the inability of the child to nurse, although obviously anxious to do so. The necessity for breath causes it quickly to relinquish the effort. This is especially marked in nasal and laryngeal affections, either obstructive or spasmodic.

Cardiac dyspnœa may come on suddenly or gradually, after exertion or when the subject has been perfectly quiet. It is often accompanied by excessive pain. The resemblance of this affection to bronchitic asthma has given it the name of cardiac asthma. The spasmodic nature and the absence of mechanical causes point to the nerve influence as an etiological factor. It not infrequently comes on suddenly during the night. Its causes have been already listed.

The peculiar sudden dyspnœa of pulmonary embolism is described in that section.

Nervous dyspnœa, the third class, has already been touched upon by reason of the impossibility of completely separating the three classes. It is apt to be combined with mechanical causes, since pressure and peripheral irritations of nerve terminals are potent factors in exciting spasm and other evidences of reflex action. Hence, in diphtheria, in all forms of laryngeal inflammation, irritations the result of of growths or foreign bodies in larynx or trachea, and, as already stated, the influence of an aneurysm, may cause alarming and even fatal spasm of the glottis. The nervous influence in the production of bronchial asthma is described in the section devoted to that malady. That it is the result of transient spasm is indicated by the suddenness with which it comes and goes, the manner in which it is influenced by

nervines, and the influence which certain odors or irritants when inhaled exercise in the development of the attack.

Hysterical dyspnœa belongs to this category, and the dyspnœa of pregnancy sometimes seen early in the case, apart from any albuminuria, cardiac or kidney lesions, is partly mechanical and partly nervous. The dyspnœa of the later stages is almost purely mechanical.

Renal dyspnœa shows itself in two forms, the earlier form usually appears after exertion and in this respect closely resembles the corresponding form of cardiac dyspnœa. The second form is probably of nervous origin, and markedly resembles spasmodic bronchial asthma. The differential diagnosis between the two forms should be based upon evidence pointing to disease of the one or the other organ. In sooth, the various forms are all differentiated by signs and evidence confirmatory of pathologic changes sufficient to account for the attack having occurred in the organs and tissues involved, and by the careful exclusion of similar changes in the other organs which might give rise to corresponding symptoms.

Orthopnœa is inability to breathe while lying down; it is a grade of dyspnœa. In some cases it is so severe that the patient must sleep with the head resting on the arms or on a table. While the patient may be able to bend forward, yet the slighest backward tilt brings a sense of impending suffocation. Orthopnœa sometimes occurs in œdema of the lungs, in high grades of pulmonary congestion and in pleural effusion, but usually it is associated with some cardiac lesion or with an advanced aneurysm. In the absence of signs of pulmonary involvement the probability of the cardiac origin is increased. Pericarditis may cause it, particularly when adhesions form. Increased strain thrown upon the heart in the course of other diseases which cause lesser grades of dyspnœa, and intercurrent cardiac complications may produce it; hence it may be paroxysmal. The assumption of the erect posture is explained by the fact that the organs are less hampered in this position, the auxiliary muscles are more easily fixed, and fluids gravitate to levels where they interfere less with the air interchange. In some cases a satisfactory explanation is not apparent.

CYANOSIS.

Cyanosis is closely related to dyspnœa, and great dyspnœa usually presents a considerable degree of cyanosis, yet each may occur without the other; thus, in that group of congenital cardiac defects

known as congenital cyanosis, or morbus cœruleus, while cyanosis is the most evident sign, dyspnœa may be wholly lacking. On the other hand, severe attacks of tachycardia may be attended with marked dyspnœa, yet cyanosis be lacking.

Cyanosis may affect the entire body, or only the extremities may show signs of it. The face is affected in all except the lighter grades. The lips and the finger-tips are the first to give evidence of it. All diseases interfering with the entrance of air into the lungs cause cyanosis directly proportional to the degree of interference. Diseases and conditions interfering with the normal exchange of gases which occurs in respiration, and which permit the retention of CO_2 within the body, naturally produce cyanosis. Hence, diminution of the cell areas of the lung, such as occurs gradually in the course of consolidations, or quickly, as in compressions and exudations, is followed by this result. Even in severe bronchitis, especially of children, a considerable degree of cyanosis is manifest. Pneumothorax exercises a similar influence. In pleurisy it increases with the amount of the fluid.

In cardiac diseases, it is one of the general manifestations, and increases with the dyspnœa. Diseases involving the right heart manifest it to a greater degree than those of the opposite side. It is marked in tricuspid disease.

Conditions of the blood which diminish the oxygen-carrying power cause lividity and cyanosis. Thus, it is seen during the administration of anæsthetics, in many forms of poisoning, as by nitrous oxide, after a dose of morphia, the inhalation of irrespirable gases, especially CO_2, and coal-gas. Diseases and neuroses of the larynx which cause dyspnœa, also cause cyanosis; as paralysis, and laryngismus stridulus of children. In the chronic infantile stridor of Taylor and Lee, dyspnœa is without cyanosis. Acute laryngitis produces lividity of lips and finger-tips. Laryngeal diphtheria may produce a high grade of general cyanosis.

The chronic cyanosis of heart diseases is followed by permanent changes of an indurative nature, as pigmentation and sclerosis. Cyanotic induration is described in connection with diseases of the heart.

CLUBBING OF FINGERS AND TOES.

In various chronic disease of the chest there occurs a thickening and curving of the finger ends and to a less degree of the toes and the nose, to which the name of clubbing is given.

It was mentioned by Hippocrates and is called "Digiti Hippocratici." Trousseau describes it as follows:

"The deformity consists in a contraction of the ungual phalanx with enlargement and thickening of the digital pulp. While the nail curves towards the palm, the extremity of the finger assumes the form of the large end of a club and sometimes in enlarging it flattens so as to resemble the head of a serpent. This deformity generally comes on by slow degrees but at other times it is produced with great rapidity, the patient suffering pain during the process. In some persons the toes are the seat of a similar deformity, but when it occurs in the toes it is generally in a much less degree than in the fingers."

Trousseau does not mention the change as affecting the nose, but other writers call attention to this fact, and I have seen it occasionally. It has also been stated that in the digital affection the nail is raised from its bed and that by bearing down on its proximal end, the hard, elevated margin of the root of the nail may be felt. While in most cases this is true, and the stretched, shining atrophic skin over the root indicates the hyperplasia beneath, yet, quite frequently in my experience, the nail has been sunken and the hyperplasia seemed to surround it as a wall. Particularly is this the case in the flat clubbing, likened by Trousseau to the head of a serpent. The enlarged digits often show cyanosis. The disease is general and bi-lateral although all fingers are not equally affected. In a recently reported case of subclavian aneurysm it occurred only upon one side. It has been known to appear and disappear without any known disease being associated. Its progress is generally slow but it may attain considerable proportions in two or three weeks. In the order of the frequency of its association it occurs in bronchiectasis, chronic pulmonary tuberculosis, especially in conjunction with cavity, emphysema, chronic bronchitis, chronic asthma, empyema, congenital heart affections accompanied by cyanosis, and certain valvular lesions. In the first mentioned, its occurrence is so frequent as to be of some diagnostic value and is apt to be associated with changes in the nose and toes. In mitral insufficiency it occurs especially when a child is the subject of the attack. Its association with pulmonary tuberculosis is well-illustrated in dispensary practice.

SECTION V.

BRONCHITIS.

Definition. An acute or chronic inflammation of the bronchial tubes occurring primarily or in the course of various diseases. When the finest ramifications of the bronchi are the seat of the disease it is called capillary bronchitis.

ACUTE BRONCHITIS.

Inspection shows only slight increase in the number of respirations, which occurs in the febrile stage of the acute attack. The respirations have a somewhat hurried character.

Palpation. Vocal fremitus normal. Occasionally a distinct bronchial fremitus is transmitted to the chest surface.

Percussion. The resonance is clear over the entire area. It may be slightly diminished in the lower and posterior regions when large amounts of mucus have accumulated in the bronchial tubes.

Auscultation. The respiratory murmur changes, becoming more bronchial and harsher in character. Expiration is usually quite noticeable, which is not generally the case in health. New sounds are present, called râles. These may at first be dry and afterwards become moist, more usually both exist at the same time and are heard in different areas. Sometimes they are so harsh in character and so widely diffused as to mask the breathing sounds. The larger tubes give rise to large bubbling or sonorous râles, the finer ones to small, hissing or sibilant râles. In a very few cases, accompanied by an extremely viscid exudate, there may be some fremitus. In these cases the respiration may be broncho-vesicular and the symptoms misleading. Ordinarily the vocal resonance is normal.

The cardinal symptoms of acute bronchitis are cough, expectoration and dyspnœa, although children below the age of five years do not expectorate, and the dyspnœa caused by a slight catarrhal inflammation of the smaller tubes may be inappreciable.

The disease generally begins as a catarrh of the nasal, pharyngeal

or laryngeal regions, with hoarseness and tickling in the throat, soon succeeded by a sense of rawness behind the sternum, and in severer cases, or in emphysematous subjects a sense of tightness in the chest.

The principal characteristics of the disease are that it is bilateral, that percussion and voice sounds are normal, that the adventitious sounds change in character and location with great rapidity, that it is accompanied by a cough that is at first short, dry, hacking, painful and which recurs in severe paroxysms. When the hyperæmia of the bronchial mucous membrane subsides and free secretion intervenes the cough loses these characteristics and becomes loose and painless, but is still paroxysmal.

The sputum of bronchitis is at first scanty, viscid and tenacious, expelled with much difficulty and adheres to the vessel. It may be blood-streaked. Later it is more mixed with saliva, less viscid, is frothy from admixed air and resembles partially-beaten white of egg. It is now scantily mixed with leucocytes. In the third stage it consists largely of an admixture of leucocytes and mucus, becomes again denser, its color changed to yellow or dirty green and the separate mouthfuls do not agglutinate. The amount may be eight to ten ounces in twenty-four hours. The microscope shows mucous corpuscles and epithelium floating in clear fluid. The epithelium is cylindrical and ciliated. In the second stage hyaline cells, leucocytes, epithelium, oil globules and a few blood corpuscles may be observed. Pulmonary phthisis is the only disease that resembles bronchitis in any of its physical signs, but the course of the disease and the absence of tubercle bacilli, soon clear up the doubt.

Capillary Bronchitis. On inspection no differences from the above signs are noted, but in advanced cases marked lividity due to lack of blood aëration is present.

Mensuration, no change.

Percussion. Note normal or slightly exaggerated.

Vocal resonance normal.

Auscultation. Vesicular murmur is weakened and in some cases suppressed. Subcrepitant râles are heard *on both sides of the chest.* If heard over considerable areas they indicate positive and extensive inflammation of the capillary tubes. These fine râles may be heard only in the dependent and posterior portions of the lung, in which case they are due to the gravitation of fluid from the larger into the

smaller tubes. Above these areas the large moist râles may be abundant or mixed with finer ones.

Differential Diagnosis. This disease might be mistaken for pneumonia, but the normal or exaggerated percussion resonance, its bilateral character with subcrepitant râles heard on both sides, and the absence of bronchial breathing, should set the physician right. The disease attacks the aged and young children. While clinically it is distinguished from catarrhal pneumonia, in practice it is often impossible to determine which disease has attacked the patient.

CHRONIC BRONCHITIS.

Presents no distinctive features, except in cases due to mechanical irritation, the so-called artisan's bronchitis. This type may be accompanied by wasting, due to induration of the lung, thus resembling phthisis, but differs from it in its essential chronicity and the absence of tubercle bacilli

EMPHYSEMA.

Definition. A chronic interstitial inflammation of the lungs, causing obliteration of the capillaries, associated with dilatation of the air spaces.

A compensatory emphysma is recognized when one lung, or a portion thereof, by reason of disease being unable to perform its functions the remainder increases in bulk and the air spaces dilate. This condition is not, however, pathologic.

Physical Signs. Inspection. The patient is cyanotic, especially after exertion. The breathing is rapid. In advanced cases the upper part of the thorax is dilated and prominent, especially the sternum and clavicles. The muscles of respiration and the elevators of the chest are hypertrophied, especially the scaleni and the sterno-cleido-mastoids. If both lungs are affected the chest is barrel-shaped, the spinal column bent forward, the shoulders rounded so that the patient seems to stoop. The middle and sometimes the upper intercostal spaces are widened, the lower ones narrowed, and the lower ribs drawn in. The above picture depicts the hypertrophic type of the disease.

There is, however, a form of the disease in which the lungs are atrophied instead of hypertrophied, but in which the symptoms do not radically differ from those of the above type. Atrophic emphysema is generally found in the aged, and may be looked upon as a part of the physiologic process of decay.

The chest movements are altered. Instead of the rhythmical expansion and contraction seen in normal respiration, the chest is lifted as though it were an inflexible piece, and, owing to calcification this is indeed often the case. The lower ribs and abdominal muscles are drawn in with inspiration, the breathing is labored and of the abdominal type; the diaphragm being brought little into action.

Palpation. Fremitus varies, sometimes it is increased, at others diminished. The apex beat is generally lowered and pushed to the right.

Radioscopy shows that in emphysema the diaphragm occupies a lower plane and does not rise during expiration to the normal level, and that the heart assumes a much more vertical position in this disease than it occupies in the healthy chest.

Mensuration. Both the circumference of the chest and the antero-posterior diameter are increased. The antero-posterior diameter often equals the transverse.

Percussion. The percussion note is clear and ringing, the pitch being lowered but the intensity of the sound increased. It is in this disease that we get the typical vesiculo-tympanitic note. The note is not changed either by forced inspiration or forced expiration. The finger feels increased resistance. The area of cardiac dullness may be absent. Although there is very generally present hypertrophy of the right ventricle, it cannot be demonstrated during life.

Auscultation. The inspiratory sound is short, feeble and even suppressed. Expiration is greatly prolonged and may be three or four times as long as inspiration. The pitch of both is lowered. Râles of all sizes and qualities may be heard. In those cases where narrowing of the bronchi has resulted from chronic inflammation, both inspiration and expiration are prolonged, nearer equal in length, harsh, sibilant, high-pitched and may be accompanied by râles, especially if, as is usually the case, bronchitis coexists. Theoretically, vocal resonance should be always diminished, owing to the lessened vibration in the air columns. As a matter of fact it varies as does fremitus, being occasionally normal or even increased without any satisfactory reason being evident.

Differential Diagnosis. Emphysema might be mistaken for pneumothorax or pleural effusion. From the first it differs in that the percussion sound is always pulmonic, even if tympanitic, and the breathing is still vesicular, while in pneumothorax both are amphoric. Em-

physema is bilateral, pneumothorax is unilateral and metallic tinkling is diagnostic. Percussion dullness over effusion contrasts strongly with the vesiculo-tympanitic note of emphysema. The malady is most frequently the sequel to bronchitis.

Hæmoptysis, although a rare occurrence in emphysema, may take place and has been known to prove fatal. In fatal cases it is probably due to rupture of the aorta, since extensive atheroma of this vessel occurs in the disease. The ordinary hæmoptysis of emphysema is small and unimportant.

SPASMODIC OR BRONCHIAL ASTHMA.

Definition. A spasmodic, paroxysmal panting for breath due to contraction of the caliber of the bronchial tubes. The seizures are probably of nervous origin.

Physical Signs. Inspection. Breathing is labored, the muscles of respiration stand out prominently, the abdominal muscles are contracted and hard. Inspiration is prolonged and labored, as is also expiration. The patient sits up, leans forward and grasps some object to aid in the fixation of the shoulder muscles. The face is cyanotic, the expression anxious. The chest is in a permanent state of inflation, hence shows little expansion with inspiration.

Palpation. Rhonchial fremitus is recognizable, vocal fremitus is lessened and often covered by the rhonchi.

Percussion is unchanged in true asthma. In associated emphysema it is exaggerated.

Auscultation. The rhythm of the respiratory murmur is jerky and irregular, now exaggerated, now suppressed. The vesicular murmur is inaudible owing to lessened vesicular interchange. All over the chest are heard sonorous and sibilant râles with both inspiration and expiration, and expiration is noticeably prolonged. The dry râles change to moist as the attack progresses and the change brings relief.

Differential Diagnosis. There are attacks of renal asthma which closely resemble the above form, but in these the dyspnœa is less marked, and the kidney lesions are manifest on examination of the urine. Rhonchi and rhonchial fremitus are less pronounced and may be absent, but the expiratory character of the dyspnœa is apparent in both forms. Spasm or paralysis of the glottis produces a dyspnœa

which resembles asthma, but in these the difficulty is wholly *inspiratory* and the râles are not present.

BRONCHIECTASIS.

Definition. An acute or chronic affection characterized by dilatation of the bronchial tubes, cough and characteristic expectoration. The dilatations may be cylindrical or sacculated. In the acute cases the former are generally found, while in the chronic cases the sacs are gradually formed. Occasionally the acute cases give rise to sacculations. There is also an acute and chronic form of enlargement of the bronchioles (bronchiolitis) but the physical signs are not sufficiently distinctive to allow the disease to be described apart from bronchiecta-

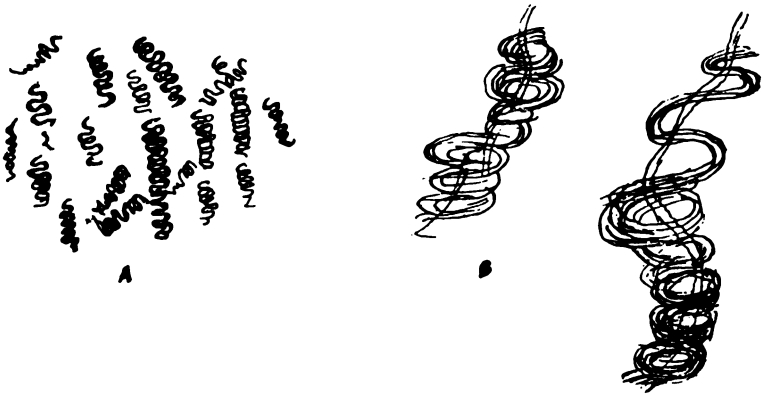


Fig. 32—Curschman's Spirals. A Natural size. B Enlarged.

sis. This latter form follows acute infectious diseases, especially measles and whooping cough. It may become chronic.

Clinically we recognize three forms of the disease:

(a) *Pure* bronchiectasis which follows after bronchitis, especially chronic bronchitis, pneumonia, pleurisy, empyema. Its association with the first is well known, but it is also especially prone to be present in lobar or broncho-pneumonia, where it is limited to the bronchi within the affected area. Influenza is responsible for not a few cases, and bronchiectasis which may be classified as acute, has succeeded its attacks.

(b) *Tuberculous* bronchiectasis. Few cases of pulmonary tuberculosis progress to the end without the formation of bronchiectasis. Sometimes the large cavities so formed are mistaken for vomicæ. In

one of my cases enormous dilatations were present in a lung riddled with tuberculous cavities.

Both the saccular dilatations and the cylindrical enlargements are found.

(c) *Traumatic* bronchiectasis, caused by the pressure of a foreign body within the bronchi or external pressure exerted on a bronchus, causing kinking or stenosis of the tube and subsequent dilatation.

In the three forms enumerated the disease is essentially chronic, cases sometimes lasting ten to twenty years. A case seen by me began with pneumonia which was followed by empyema and then by bronchiectasis.

The physical signs vary with the location, size of the sac and the amount of its contents, giving rise to wholly different signs when full, partly full or empty. The sacculated cases are unilateral. The cavities most often form in the lower lobe. They are rarely single, sometimes two communicate by a narrow passageway. Oft times there are two large ones and one or two of lesser size. The size of the dilatations varies greatly.

Inspection shows deficient expansion over the affected area. Breathing is labored, expiration prolonged. The expression is anxious, varying degrees of cyanosis are not uncommon. In chronic cases the fingers are clubbed, the nails incurved. In cases following chronic lung diseases, especially an old empyema, a pneumonia, or a pleurisy where, as is so often the case, adhesions have followed, the retracted side, the compensatory bulging opposite to it, the shoulder droop on the affected side, and the forward curve of the spine form a most impressive image. If upon these is engrafted, as is occasionally the case, a hyperostosis of the extremities, hands, fore-arms, feet and legs, the picture is ineffaceable. This proliferating hyperostosis, described by Marie and named osteo-arthropathie pneumique, occurs in other chronic lung diseases, and differs from acromegaly in that it affects only the parts mentioned and is a true proliferation.

Palpation. Vocal fremitus is usually increased by reason of the associated induration resulting from the causative disease, such as broncho-pneumonia. Rhonchial fremitus is also present. The fremitus may not be manifest unless the voice is considerably raised.

Percussion. There is very generally a considerable area of dull percussion resonance, even of flatness, in the region affected, which is

more often the lower lobes. Dullness will be found posteriorly and laterally oftener than anteriorly.

With a full sac surrounded by induration *flatness* prevails. With a large, completely empty sac the note is tympanitic. With smaller sacs and with cylindrical enlargements the note is tubular.

Auscultation. The breath sounds are absent with a full sac. They are bronchial, cavernous or amphoric when the sac is empty. When half-full splashings, succussions and bubblings are heard with both inspiration and expiration. With an empty sac, bronchophony and pectoriloquy may be heard.

When the cavities are full breathing may be deficient over the entire affected half of the thorax, but is subject to rapid changes. A full inspiration, a cough or even a change of position renders it audible.

Râles. Extensive râles are heard *par tout*, large, loud, sonorous, gurgling, mucous, rapidly changing. They are especially manifest and characteristic when the cavity is partially filled.

Cough. The patient may have considerable intervals of freedom from cough, days or weeks. This and the character of the cough render it distinctive. It is always paroxysmal, usually matinal, the patient generally having a premonition of its oncoming. Each paroxysm may exhibit a descending scale both as to the intensity and the force of the succeeding notes. Thus, a single paroxysm may consist of six notes, the first loud, hard, expulsive, with each successive note a little less intense than its predecessor. After a brief pause a repetition of the effort occurs, until with some chokings and gaspings, the cavity is emptied.

Sputa. This varies in amount with the size of the cavity and the frequency with which it is emptied. Four drams to half-a-cupful is an ordinary amount. Its color varies: white, gray, yellow, green, brown. It is exceedingly offensive, the odor being fœtid or cheesy, "as of the ripe Camembert cheese."

When placed in a tall glass the sputum separates into three layers. An upper, frothy layer which may be colored, a middle layer of watery mucoid fluid; the third, a thick sediment of granular matter, cells, casts of the tubes, bacteria, crystals of fatty acids, and, if ulceration of the walls be present, elastic fibers are found. Sections of the casts show peculiar alternate layers of inspissated secretion and bac-

SECTION VI.

PLEURISY.

Definition. Pleurisy is an inflammation of one or both pleural sacs. Varieties. Primary, secondary: Acute, chronic: Circumscribed or diffused: Dry pleurisy, pleurisy with effusion.

Because this disease so often passes unrecognized by the physician, in spite of its pronounced physical signs, as evinced by the fact that extensive adhesions are often found post-mortem without any history of pleurisy being elicited, it deserves a minute description.

The various stages of the malady present different signs, and will therefore be considered separately. Inspection, palpation, mensuration, percussion and auscultation all give intelligent answers when applied as interrogatives to this disease.

In the beginning of an attack, whatever form it may afterwards assume, the lesion is that of a *dry* pleurisy, in which the two contiguous layers, first the visceral and then the parietal, become inflamed. Following this an exudate of thick, soft gray or yellow lymph is deposited upon the two surfaces, sometimes in distinct layers. At first this lightly adherent lymph may be easily rubbed off, after a time it is sticky, rough, shaggy and quite adherent. The two surfaces may now become agglutinated and the attack thus terminate (Pleuritis sicca).

In the beginning and at its height such a condition presents the following signs:

Inspection. Increased frequency of breathing but with restriction of the respiratory movements. Enfeebled respiration which is made manifest by comparison with the sound side. The limitations of the respiratory excursion vary with the severity of the pain.

Palpation. Touch will sometimes reveal a peculiar fremitus caused by the friction of the two surfaces (pleural fremitus of Da-Costa) over the region corresponding to the area of inflammation. Vocal fremitus during the inflammatory stage remains unaltered or is even increased over the inflamed area. Absent vocal fremitus is one

of the early signs of effusion. The reader is again reminded that vocal fremitus is normally more marked on the right side than on the left.

Mensuration. Previous to the exudate, no change. (Right side normally the larger.)

Percussion. Prior to the effusion percussion elicits little or no information.

Auscultation. In the presence of severe pain the respiratory rhythm changes and becomes irregular and jerky. Likewise pain causes the respirations to become shallow or enfeebled, in which case the normal vesicular murmur is diminished—thus confirming the signs elicited by inspection. Friction sounds are audible especially on deep breathing. They are superficial and have a sandpaper quality. Generally they are heard on both expiration and inspiration; sometimes they are limited to expiration. They may be limited to a single spot in the inframammary or infraaxillary space, and hence be easily overlooked. Usually the area is more extensive. The sound resembles the crepitant râle, but this râle is an *inspiration* râle.

PHYSICAL SIGNS OF EFFUSION.

Inspection. With moderate effusions inspection shows lessened chest movements, due to inability of the lung to expand to its full capacity. Pain may or may not be present. When present it is usually accompanied by an increase in the number of the respirations, which may even amount to dyspnœa. In the absence of pain, respirations may be even lessened in number. The interspaces above the diaphragm may be widened. A moderate effusion is one in which the level of the fluid does not rise above the nipple in front.

Palpation in moderate effusions. Confirmation of signs revealed by inspection, as diminished respiratory rhythm and widening of the lower interspaces. The friction fremitus will have disappeared. In small effusions the tactile fremitus is generally diminished. This sign is more valuable in men than in women and children. It must be remembered that fremitus is usually stronger on the surface of the right chest than on the left; stronger in men than in women.

Mensuration shows little if any variation in small and moderate effusions. In right-handed individuals, especially men of the laboring class, the circumference of the right chest is normally greater than that of the opposite side.

Percussion. Dullness increasing to flatness over the area of the

effusion is the most important sign of the exudate. In adults it is difficult if not impossible to demonstrate effusions of less than five hundred cubic centimeters. The dullness is usually most marked and first obtainable posteriorly, then in the axillary region, lastly in front.

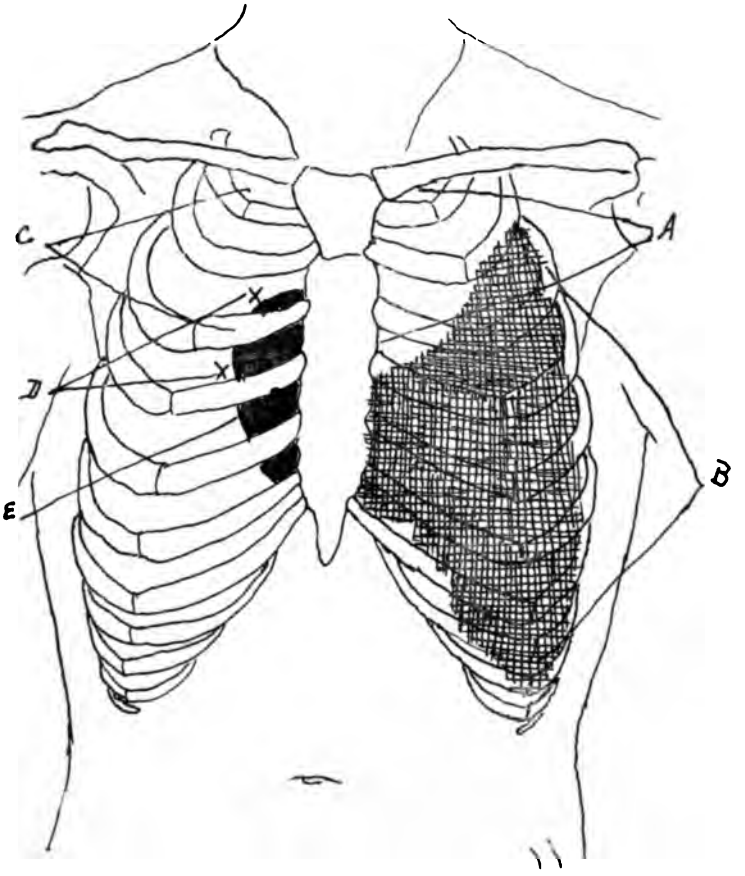


Fig. 33—Physical signs in effusion into left pleura (Patton). A Skodiac percussion resonance, exaggerated vocal resonance, tubular quality of breathing. B Complete dullness, absence of vocal fremitus, no respiratory sounds. C Purile respiration, prolonged expiration, exaggerated percussion resonance. D More or less well marked pulsation. F Displaced area of cardiac dullness. Upper limit of shading indicates anterior line of Ellis' curve.

Along the upper border of the effusion percussion dullness is obtained and increases to flatness as we percuss from above downward. Total absence of vibration, coupled with an increased sense of re-

sistance under the pleximeter finger, much greater than that observed in cases of lung consolidation, are most noteworthy and significant symptoms.

The prevalent, in fact almost universally accepted, belief that the level of pleural effusions changes with alterations in the position of the patient is almost wholly erroneous, and is the probable reason why such effusions frequently are overlooked. Only occasionally does such change of level occur, and I wish to lay special stress upon this fact.

As pointed out by Flint, Da Costa, and others since, we get increased percussion resonance, even amounting to tympany, above the level of the fluid. This peculiarity is due to disturbance of normal tension in the pulmonary tissue, which is more or less condensed. The change of quality is variously known as Flint's vesiculo-tympany, Skoda's resonance, etc. In moderate transudations the upper level of the effusion, and hence of percussion dullness, is not a straight line but a horizontal curve with the convexity upwards. The curve reaches its greatest height in the mid-axilla, whence it descends to the sternum, where, however, the level is higher than at the vertebra. In order to demonstrate this curve the patient should be in the upright position. In a moderate effusion this line may extend from the junction of the sixth rib with the sternum, which point is one inch above the xiphoid junction, to the lower border of the fourth rib in the anterior axillary line, thence slope gently backwards. This line of dullness does not change with inspiration or expiration but *may* change with changes of position.

Auscultation. Moderate effusions. The breath sounds become feebler and more distant as the amount of fluid increases. As has been stated, the vesicular portions of the lung are pressed upon and egress of air is prevented, hence vesicular respiration is absent and is replaced, first, by broncho-vesicular and then by bronchial breathing. In children this occurs very early and with comparatively small effusions. Sometimes the breathing is amphoric. On the opposite side the lung is doing increased work and the breath sounds are therefore intensified or puerile.

Vocal resonance varies. When the effusion is small in amount the voice is still conveyed *via* the chest walls to the ear. With increased effusion the voice sounds are absent over the liquid. If listened to at the upper border of the fluid a peculiar quavering or bleating, called egophony, may be heard. It is not often present, but

when heard is decisive. In typical forms egophony is uncommon, although much insisted on by the older writers, but it is by no means rare to hear a curious nasal, twang-like quality in the voice, particularly at the outer angle of the scapula.

Moderate effusions, while they compress the air cells do not affect

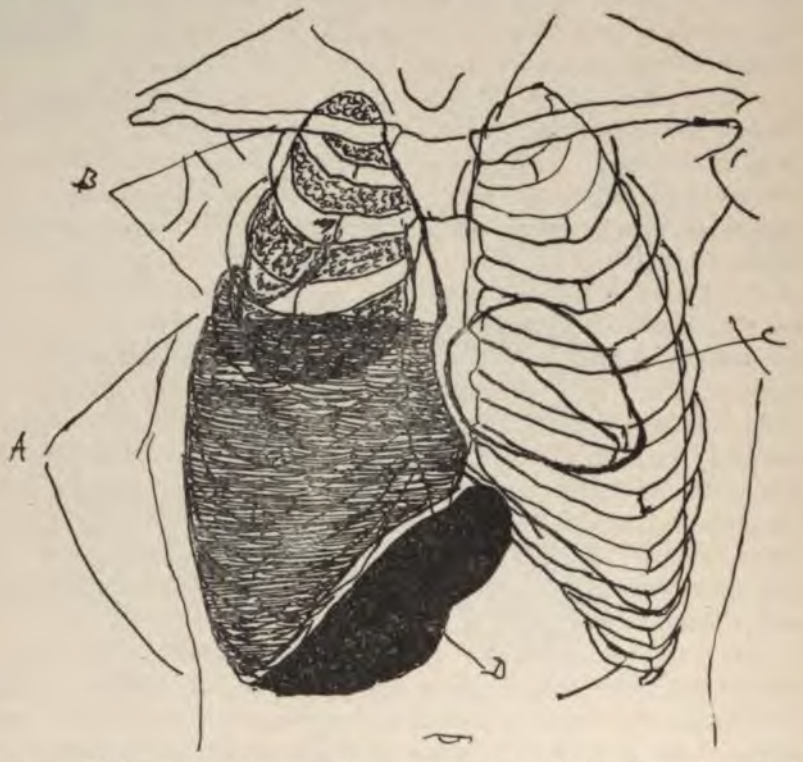


Fig. 34—Displacement of organs in a considerable effusion into right pleura (Patton). A Slight enlargement of side, with effacement of intercostal spaces. B Compressed lung. C Displaced heart. D Displaced liver.

the bronchi. Bearing this fact in mind aids in comprehending many of the observed phenomena.

LARGE EFFUSIONS.

Inspection. The respiratory movements are much diminished or entirely absent on the affected side, and increased on the sound side. The intercostal spaces are widened, especially in the lower segment. Even bulging may be noted. The affected side looks inflated, the

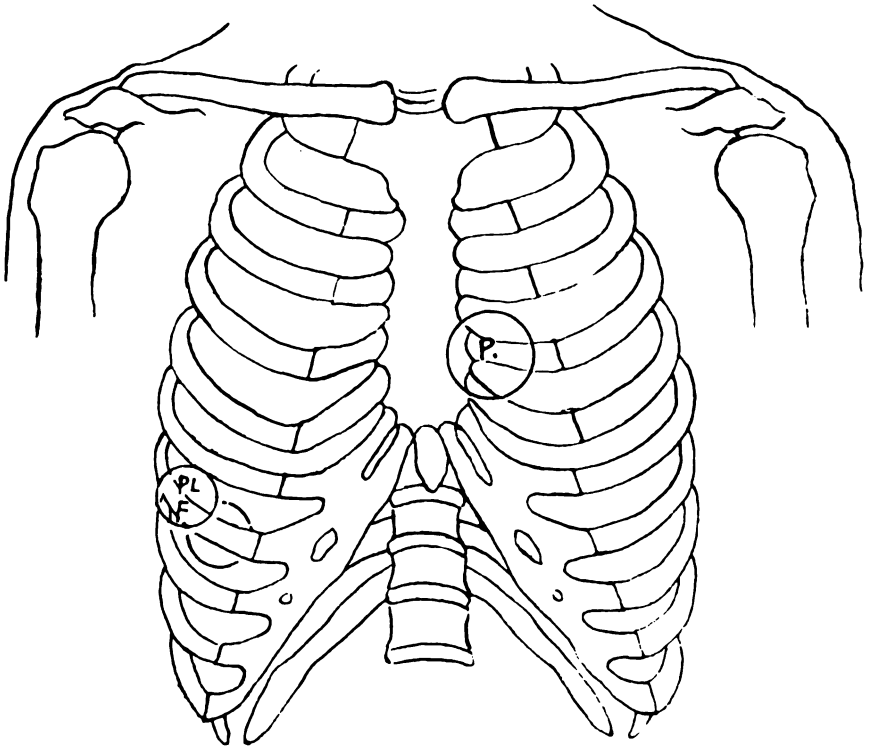


PLATE IV.

P. Point where Pericardial Friction is most often heard. PL.F. Point where Pleuritic Friction is most apt to be heard. The Inner Circle represents the most frequently affected Dorsal Area.

scapula stands out more prominently than its fellow, the shoulder is elevated.

The heart impulse is moved to the left in right-sided pleurisies and is found outside the nipple line. It is sometimes elevated slightly. In left-sided pleurisies the apparent displacement is much greater, and the apex impact may be found a considerable distance to the right of the sternum, even in the right nipple line.

Palpation in large effusions confirms inspection as to lessened movement on breathing, widening of the interspaces and abnormal position of the apex. The impact of the heart apex in disease, as in health, often may be felt when it cannot be seen. Tactile fremitus is absent over large infusions, but in children even with a considerable transudate, both vocal and tactile fremitus may be present, a point not to be forgotten. In these cases the sound probably reaches the ear through the medium of the chest wall.

Mensuration. The affected side is larger than the sound side. The difference seldom exceeds an inch or an inch-and-a-half. A cyrtometer traging of the two sides shows marked differences in the contour.

Percussion. As the level of the fluid rises it is followed by the dull percussion note, which changes to flatness from above downward. Flatness is generally first demonstrable posteriorly at the base of the lung. Resistance under the finger and absence of communicated vibrations which only occurs in the presence of confined fluids in the chest, are signs whose value have been previously emphasized. Except in effusions which absolutely compress (carnify) the lung, tympanitic resonance may be obtained in front, beneath the clavicle and above the spine of the scapula posteriorly. The cracked-pot sound may be elicited by percussion below the clavicle, while the patient expires with the mouth held open. Below the fluid level absolute flatness extending to points lower than the normal limits of the lung is present.

Auscultation. Vocal fremitus is absent, but Baccelli has noted that the whispered voice sometimes may be transmitted to the ear when the spoken sounds are inaudible. He states that this is not the case if the fluid be purulent, hence this sign, if present, denotes serous effusion. If the effusion is sufficiently large to squeeze the lung upward and backward into the spinal gutter, then the breath sounds wholly disappear. Usually, however, some breath sounds, feeble and distant, but of a bronchial character, may still be heard over the spinal

gutter when the patient folds the arms tightly over the chest, so as to widely separate the scapulæ. Bronchophony is heard in the same region.

Sometimes auscultation will enable us to locate the apex of the

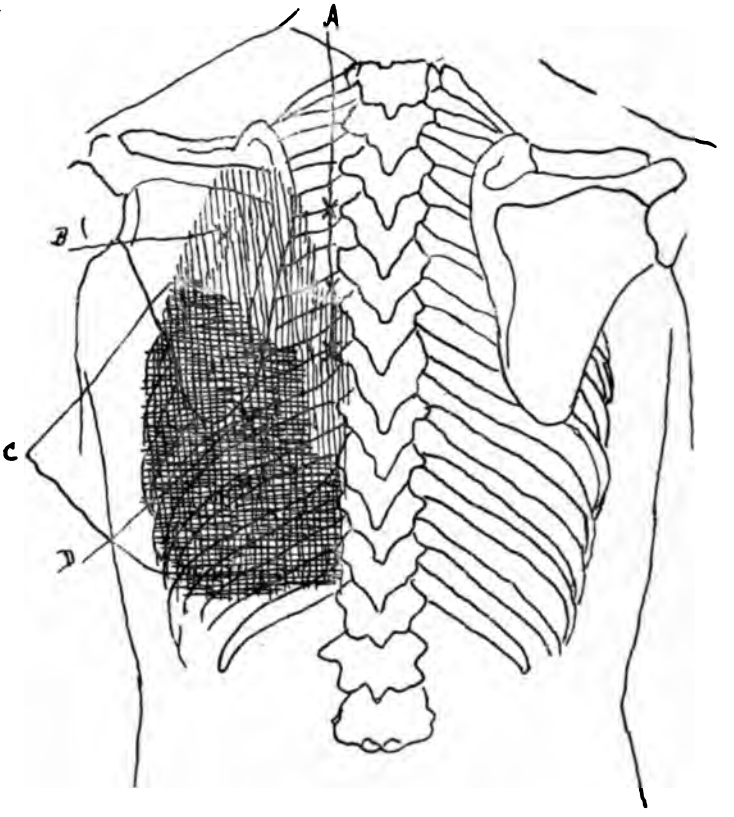


Fig. 35—Physical signs (posterior regions) in effusion into left pleura (Patton). A Diminished respiratory sounds. B Very faint or absent voice sounds, absent breath sounds, dullness, no vocal fremitus. C Dullness, no vocal fremiitus, no voice or respiratory sounds. D Voice sound is tubular or nasal in quality, sometimes approaching ægophony. Upper limit of light shading indicates the posterior line of Ellis' curve.

heart when it can neither be seen nor felt. On the opposite side the respiratory sounds are exaggerated.

Absorption Stage. Inspection shows that the enlargement of the affected side is disappearing, that the widening interspaces are re-

turning to their normal condition. The absent respiratory movements return, and the obscured apex may now be seen.

Unfortunately the process does not generally stop here, but continues by reason of contractions and adhesions until various deformities plainly show the ravages of the disease. The organs may be permanently displaced. Owing to adhesions the slow contraction of the connective tissue thus formed, and the organization of the exudate, the resulting deformity appears gradually, after a considerable interval, and is progressive. In children it may entirely disappear with growth. In adults it is permanent and varies from a hollow depression in the ensiform region to antero-posterior flattening of the chest, flattening and retraction of its lateral areas, the lower ribs being in contact or overlapping, and spinal curvature of greater or less degree.

Palpation shows a gradual return of fremitus, vocal and pleural, and a return of the organs to their normal position.

Percussion. The return of normal percussion resonance is gradual, returning first above and extending downward. At the base resonance may remain permanently absent owing to accumulations of solidified plastic material or to solidification of lung tissue.

Auscultation. Respiratory sounds return. At first they are weak and distant but increase in distinctness until they have even a harsh quality. With disappearance of the effusion, a return of the friction sound is noted. At the base of the lung all sounds may be feeble and obscured for a long time. As the vesicles gradually distend a peculiar creaking or crackling sound is heard. Bronchophony reappears and in some cases egophony.

In cases where a portion of the lung remains permanently impervious, there is permanent loss of respiratory motion and of vocal and respiratory sounds over such areas.

DIAGNOSIS.

As said in the beginning of the section, pleurisy passes unrecognized perhaps oftener than any other disease occurring within the thorax. In the early stage the friction sounds may be absent and this absence is one of the chief misleading factors. The disease is not to be mistaken in the first stage for pleurodynia or intercostal neuralgia. Both of these disorders are unattended with pyrexia, the tenderness is superficial and excited by irritation of the skin. Pain in pleurisy is deeper-seated. The pain of pleurisy may however be re-

ferred to points distant from the seat of the lesion. Thus, the seventh, eighth, ninth, tenth and eleventh thoracic nerves after passing through the intercostal muscles at the side of the thorax, so as to *lie against the pleural sac*, afterwards pass between the obliquus internus and transversalis muscles and ultimately reach the skin of the anterior *abdominal* wall. Hence the frequency with which pain is referred to these terminals, a fact which should be borne in mind, and an examination made along the entire course of the nerves seemingly involved. In double pleurisy the pain is referred to symmetrical points on the thorax or abdomen, but in general symmetrical pains are more probable indications of spinal disease. The pleural friction sound is generally audible with the beginning of expansion, differing in this respect from the fine crepitant râles of pneumonia.

Left-sided pleurisy may be mistaken for pericardial effusion and *vice versa*. In addition to the description given under pericarditis, the following points should aid in the differentiation:

Pericarditis.	*Left-Sided Pleurisy.*
Apex impact usually cannot be seen nor felt. Heart sounds feeble and distant.	Heart apex is displaced to right. If much displaced, probability of effusion is increased. If not displaced effusion is improbable. Apex impact visible but displaced. Sounds not changed in intensity.
Dullness extends upward in pear-shaped area.	Dullness follows around the axilla in usual line.
Vocal fremitus present. Tubular breathing when great compression of the lung.	Vocal fremitus absent. Breathing sounds absent; if tubular, strongly marked.
Orthopnœa. Faint cyanosis of face and extremities.	No cyanosis, no orthopnœa.
Rotch's sign, absence of resonance in right fifth intercostal space.	Rotch's sign absent.

In the pre-exudate stage the location of the friction sound, its superficial character and its relation to the systole and diastole of the heart, differentiate the two conditions.

The effusion may be mistaken for pulmonary consolidation, either

of pneumonia or tuberculosis. It is possible also to confound cancer of the lung and various enlargements of the spleen with the effusion of pleurisy. From pneumonia and tuberculosis it is distinguished by the increase in size of the affected side, widening of the intercostal spaces, absence of vocal resonance and of fremitus. In the former disease the percussion sound is dull, in pleurisy it is flat, and in neither of the former does the dullness follow Ellis's curve. The bronchial breathing of a lung partially compressed by pleural effusion is much deeper-seated, more diffused and usually unaccompanied by râles. In pulmonary tuberculosis the disease generally progresses from above downward, while in pleurisy, as pointed out, the reverse obtains. Pleurisy is unilateral. Tuberculosis sufficiently extensive to simulate it would be bi-lateral. The history further aids the diagnosis.

Enlarged spleen, if it disturbs the position of the heart, raises it instead of pushing the heart to the right. It does not interfere with the respiratory sounds of the left side, nor cause bulging of the intercostal spaces. Posteriorly the percussion sounds are normal at the pulmonary base, where they should be first affected in pleurisy. (See also Pneumonia for further differences.)

The urine in pleurisy presents a marked contrast to that of pneumonia. While the organic solids are increased and the volume diminished, yet the *chlorides*, sulphates and phosphates are but little changed and albumin is very rare. Peptone is often present during resolution and indicates absorption of the exudate.

PNEUMOTHORAX.

Definition. The presence of air in the pleural sac. Since air is almost never present unassociated with an effusion, either serous or purulent, we therefore have hydro-pneumothorax or pyo-pneumothorax, as the case may be.

Air may find entrance into the sac from perforation of the parietal layer of the pleura or from perforation of the visceral layer. The latter is much the more common and the largest majority of all cases results from tuberculous ulceration. Emphysema probably stands second as a cause. The left side is more often affected than the right. The organs are then displaced to the right and the lung may be collapsed and pressed against the spine as in pleurisy.

The amount of associated effusion varies. Some cases are not discovered until post-mortem examination is made.

The *Physical Signs* vary considerably according as to whether air alone is present or whether it is associated with effusion.

Inspection reveals asymmetry with enlargement and immobility of the injured side, this half of the chest being in the position assumed in full voluntary expansion. This is easily demonstrated by noting the resulting symmetry on causing the patient to forcibly inspire, the asymmetry returning on forcible expiration. Bulging of the intercostals may be present and it is to be noted that there is no recession of the spaces on inspiration. This is especially the case if the opening into the pleura is valve-like so that there is actually increased inter-thoracic air pressure, as is often the case. The heart impulse is generally displaced to the right. The costal movements of the opposite side are increased. Vocal fremitus is diminished or absent.

Mensuration. The tape shows marked increase in the measurements of the affected side, and little or no change with expiration.

Percussion elicits a tympanitic resonance over the whole of the expanded side. The note varies with the amount of air imprisoned within the cavity and the degree of tension under which it exists. It may be vesiculo-tympanitic if little or no pressure is exerted or it may be purely tympanitic, indicating tension and pulmonary compression. Often it is amphoric with a rich metallic quality. The pitch varies with the conditions named and deserves especial mention. If the ingress and egress of air are free the pitch is low, and may be raised by causing the patient to inspire deeply and hold the mouth closed. From this pitch the note rises with the pressure until flatness is reached. When the air is confined under such extreme pressure that vibration from percussion is impossible, the resulting note must be flat and toneless. A lesser tension elicits a very high-pitched percussion note. Percussion of the lower areas of the chest yields dullness owing to the presence of fluid, but it is to be noticed that the tympanitic areas often extend much beyond their normal fields. When fluids are present the flatness elicited by percussion changes with changes in the position of the patient. This variation of the percussion level occurs much more readily and frequently than in pleurisy, where it is often impossible to demonstrate any such change.

Auscultation. Absence of all vesicular murmur is noticed. Such breath sounds as may be occasionally heard are feeble, indistinct and distant and are never vesicular, but rather amphoric in character, or,

as occurs in pleurisy when the pressure is such that the vesicles are obliterated, we get bronchial breathing. The vesicular murmur on the sound side is intensified and approaches puerile breathing. The vocal resonance is amphoric and may be imitated by speaking softly into a large bottle. Râles are generally present and assume peculiar

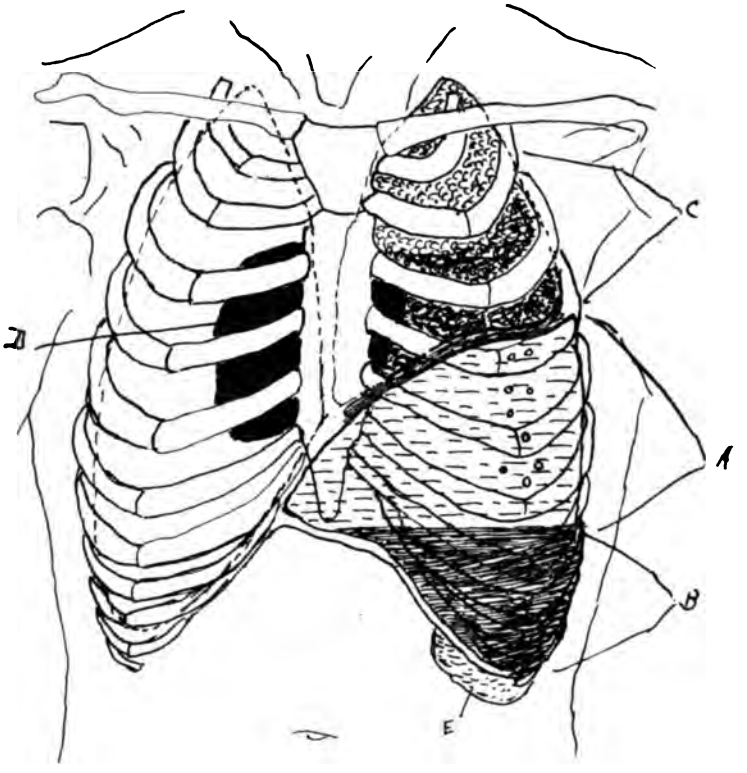


Fig. 36—Physical signs in pneumothorax (Patton). A Tympanitic percussion note, breathing suppressed, tubular or amphoric, voice sounds metallic or amphoric; metallic tinkling. B Dullness, absence of all sounds. C Harsh breathing (upper portion), increased percussion note and increase in pitch of voice and respiratory sounds (lower portion). D Displaced area of cardiac dullness. E Displaced spleen.

characters. One very characteristic sound is known as metallic tinkling and may be likened to drops of water falling against a very thin wine goblet, or to striking the goblet with a delicate wire. These phenomena were first described by Laennec, but his explanation of the manner of their production is of doubtful correctness.

Trousseau describes a valuable sign. Place a coin over the affected side and strike it sharply with the edge of another coin while the ear is held against the chest of the patient. A bell-like ringing or tinkling is heard which is pathognomonic, and may be obtained even when intrapleural tension is so high as to yield percussion dullness.

Succussion splash may be elicited by violent oscillation of the patient while the ear is applied to the chest wall. The patient should be in a sitting posture. Sometimes the patient himself is aware of this phenomenon. It even may be felt by superimposing the hands over the ribs, one on either side, while the patient quickly moves the body. This sign, which is known as Hippocratic succussion, is fully described under Auscultation in the introduction. It requires for its production the presence of both air and fluid in a cavity. When present its diagnostic significance is absolute. No one should be misled by the possible splash of a stomach partially filled with liquid contents.

In pneumothorax the organs are often displaced, especially the heart and liver. The first may be pushed either to the right or to the left; the latter displaced downward.

SECTION VII.

PNEUMONIA.

LOBAR PNEUMONIA.

Definition. An inflammation of the lung substance accompanied by exudation from the blood vessels and the growth of pathogenic bacteria, being due to a specific infection.

The lower lobe of the right lung is most often the seat of the disease, and, next, it most frequently attacks the lower left lobe. The lower lobes are affected nearly three times as often as the upper lobes.

The physical signs vary with the stages of the disease, which are called, respectively, stage of congestion, stage of red hepatization, and stage of gray hepatization.

FIRST STAGE.

No consolidation has yet taken place although there is some exudation into the air vesicles.

Physical Signs. Inspection. The patient may lie on the affected side, or be propped up, leaning toward, or "favoring," that side. Dyspnœa at this stage is not marked, orthopnœa is not frequent. The movements of the affected side are more or less restrained.

Palpation. On touch the fremitus is normal.

Percussion. The percussion note is at first unchanged but as the air spaces are trespassed upon the note becomes higher in pitch, its duration shorter and its approaches to dullness in direct proportion to the amount of exudate into the air spaces.

Auscultation. Very early in the disease, even before exudation has taken place, the respiratory murmur diminishes in intensity over the affected area and becomes correspondingly exaggerated in other parts. This quiet, suppressed breathing in one part of the lung with exaggeration in other parts, especially on the opposite side, is quite marked even in the preliminary stages and should always suggest pneumonia. Likewise during this stage of enfeebled respiration, after exudation occurs, on drawing a long breath the sounds over the in-

disposed area will be observed to have that harshness of quality and elevation of pitch to which the name *broncho-vesicular* breathing is applied. As soon as exudation takes place the crepitant râle is heard, the distinctive sound of the first stage of pneumonia. This râle is a fine, crackling sound heard close to the ear, occurring at the end of inspiration and may not be audible until a full breath is drawn. Opinion differs as to whether this crepitus is due to a fine exudate upon the pleura or is caused by infiltration into the air cells and finer bronchioles, due to the separation of the sticky exudation with inflation of the cells. While *a priori* the latter view seems more reasonable, yet it is probable that the former is correct. Coarser râles may be present in the large bronchi and may mask the more delicate crepitant sound. Moist râles are seldom present at this stage.

An examination of the urine will show diminution of the chlorides and perhaps albumin will be found.

SECOND STAGE.

Inspection. The restricted movement of the affected side is now marked and the compensatory or exaggerated breathing on the opposite side may attract attention even before the patient is uncovered. The respirations are increased in frequency and are of a labored character, as indicated by calling into play the accessory respiratory muscles. The nostrils dilate on inspiration, always a sign of *inspiratory* dyspnœa. The area of the cardiac impact may be much larger than normal, as pointed out by Graves, especially in pneumonias of the left upper lobe. The cardiac impulse may be transmitted through the solidified lung so as to cause marked movement of the chest wall.

Mensuration. The affected side is increased in volume though the increase is slight and not sufficient to obliterate the intercostal spaces. (Contrast with pleurisy.)

Palpation. Vocal fremitus is generally increased over the consolidated area, as is to be expected. Yet for some unexplained reason it is sometimes diminished or absent. This diminution may be due to the closure of the larger bronchi by the exudation, thus preventing the voice sounds from reaching the periphery. In such cases a vigorous cough may clear away the obstruction and restore the fremitus. Lack of expansion may be more apparent to touch than to sight, especially by a comparison of the two sides. Friction fremitus often may be felt. (See pleural fremitus.)

Percussion. There is marked percussion dullness over the area of infiltration. The note varies with the degree of distention of the air spaces. Sometimes the note is almost tympanitic, or at least suggests that quality, and varies therefrom to marked dullness, but never reaches that absolute flatness which has been described as the distinctive quality of effusion. Where consolidation is central and surrounded by distended, pervious tissue, we get the tympanitic note, and here even deep percussion may not elicit dullness. In the aged especially must we be on our guard against variations and irregularities. During this stage sometimes percussion elicits the cracked-pot sound. (See Phthisis.)

Auscultation. The crepitant râle appears with the exudation upon the pleura. It may persist through the second stage, but sometimes is absent altogether.

The respiratory sounds are propagated from the large bronchi through the consolidated tissue and have therefore the quality of *the tubes which remain pervious.* The breathing is therefore tubular, much intensified and is similar in quality to that heard when in health the stethoscope is placed over the roots of the lungs or over the large bronchi. Breathing may be more intense and bronchial in this disease than in any other pulmonary condition. Râles may or may not accompany this blowing respiration.

From the above explanation it follows that in cases where the large bronchi are closed by exudate, the bronchial respiration is also absent. Bronchophony is generally heard when bronchial breathing is heard, but if consolidation is imperfect it may be absent. It may have the bleating or nasal quality to which the name egophony is applied.

THIRD STAGE.

Gray Hepatization. Movements gradually return and respiratory harmony is established. Respirations become less labored and less frequent as permeability returns. If there has been enlargement of the affected side, it disappears. With increased motion of the lung the crepitant râle may be manifest.

As softening progresses air enters the vesicles, but the tubes are loaded with the softened products and râles are heard over all parts, coarse and bubbling as well as finer, dryer sounds, crepitant and subcrepitant. The name râles redux has been given to this process.

Bronchial breathing and bronchophony disappear and are replaced by the gentle vesicular rustle. Last of all to disappear is the dull percussion note and the cracked-pot sound. Impaired resonance over certain areas may permanently remain.

It will be seen that the third stage repeats largely the inspection, palpation and auscultation signs of the first stage in inverse order. The third stage, which is the stage of resolution, occupies ten or twelve days. The last sign of this stage is the crepitant râle, the return of which is known as "crepitans redux." When it is replaced by normal vesicular breathing, dullness should have disappeared.

Besides recovery and death, two other terminations should be noticed, abscess and gangrene. Osler asserts that ordinary fibrinous pneumonia never terminates in tuberculosis, that such supposed cases were tuberculous from the outset.

Abscess results from pneumonia in about three or four per cent. of the cases. Small cavities may unite into one involving a considerable portion of the lobe. The sputum becomes abundant, purulent, contains elastic tissue and may contain crystals of cholesterin or of hæmatoidin.

The cough comes on in severe paroxysms, with profuse expectoration. The fever is at first remittent, then intermittent and hectic. The signs of cavity are not usually present.

Gangrene occurs about as often as abscess, but may present no signs except post-mortem. It may occur with abscess. If the sputum becomes fœtid and exhibits the characteristics of gangrene, the diagnosis is clear.

Special Symptoms. Herpes develops in about one-third of all cases. Coming on early it is of decided diagnostic value as it appears in scarcely any other respiratory disease, although it occurs about as frequently in malaria and cerebro-spinal meningitis as it does in pneumonia. The commonest seat of development is about the angle of the mouth and nose. Less usual seats are about the eye, ear, the genitals or the anus. The vesicles appear in a crop of half a score to a score, disappearing in a few days, leaving behind dry scabs.

Chill. In no other disease except malaria is chill so apt to be an initial symptom. Often it comes on at night and is always severe, lasting fifteen to forty-five minutes. It is followed by rapidly rising fever which runs a course between 100° and 105°, F., most frequently falling by crisis between the third and the eleventh day.

Pain. Most cases are ushered in by sharp, lancinating pain, which is due to the accompanying pleurisy and therefore has its usual seat over the affected side, yet the pain often appears before the pleuritic friction is observable and may be referred to some distant point. Pain is generally most marked in the mammary or the axillary region. In old persons it is less distinct or absent. In central pneumonias it is absent. It is often referred to the abdomen in children.

Pulse varies with the fever but rises to 100° or 120°.

Dyspnœa occurs early and is severe. The respirations quickly reach thirty; later fifty or sixty in the adult is not uncommon; eighty is not extraordinary, especially if both lungs are involved. In children one hundred is not unknown. The movements are restrained on account of pain, hence are shallow, often ending in an expiratory grunt. Deep breathing elicits exquisite pain. The respiration-pulse ratio is more significant in pneumonia than in any other disease, reaching one to two or one to one-and-a-half in some cases, instead of the customary ratio of one to four. Thus, a pulse rate of 100 or 110 may coincide with a respiration rate of 50.

The *cough* is very painful and therefore restrained. It is at first short, frequent but single, dry and hard. Later it is accompanied by characteristic expectoration. In children and in old people the cough may be slight and attract little attention.

EXPECTORATION.

The first expectoration consists of small, glairy mucus, arising from the concurrent bronchitis. Within twenty-four hours after the initial chill the sputum assumes distinctive characters, becoming exceedingly tenacious, viscid and gelatinous or gluey, then tinged with blood. The cup may now be inverted without spilling the contents. The mass trembles like jelly. The patient has difficulty in expelling the sticky masses. From being blood-tinged, it becomes decidedly colored and the designation "prune-juice sputum" is sometimes applicable, though, in my experience, infrequently in frank pneumonias. In cases of the asthenic type it is common. The term "rusty" better describes the secretion in the majority of instances. The amount varies from one to four ounces in the twenty-four hours. After the crisis the color changes to greenish, then yellowish purulent masses are expectorated and gradually it fades away. Sometimes after crisis it ceases suddenly.

Microscopically the sputum is found to contain degenerated bronchial and alveolar epithelium, mucus, leucocytes, blood corpuscles changed and unchanged, minute fibrinous casts of the capillary bronchi and alveoli, hæmatoidin crystals, the micrococcus lanceolatus and sometimes other organisms, such as Friedländers bacillus pneumoniæ, staphylococcus and streptococcus pyogenes and various others.

BACTERIOLOGY.

Owing to the confusion which is apt to exist in the mind of the student with regard to the bacteriology of pneumonia the following statements are made:

Friedländer in 1882 described capsulated bacilli as being of constant occurrence in the alveolar exudate. They were named "Friedländer's bacillus pneumoniæ." He believed they were pathogenic. Frankel and others subsequently showed that this bacillus was of comparatively rare occurrence in pneumonia, and that another, discovered by Sternberg, was present in the sputa of nearly all cases. Friedländer's bacillis is now named, "The pneumo-bacillus."

This second organism has been known by various and confusing names and is still called the diplococcus pneumoniæ and also the pneumococcus of Frankel.

In 1888, Klein described yet another bacillus, known as Klein's bacillus pneumoniæ.

In some cases of the disease only streptococci are found. Frankel's pneumococcus has been found in the saliva and bronchial secretions of healthy individuals. In some cases of pneumonia none of the above organisms is present.

The diplococcus pneumoniæ, also called the micrococcus lanceolatus, and pneumococcus, is oftenest found in the sputum, but in the absence of physical signs and clinical symptoms does not indicate the presence of the disease, since it may be found in healthy persons.

Method. Smear two cover glasses with the sputum, rub together into a thin layer, separate, dry in the air and fix by passing a few times through the flame of an alcohol lamp. Immerse in a one per cent. solution of acetic acid for two minutes, draw off the excess of acid with a pipette and stain, first in a saturated solution of anilin water, then in gentian violet. Wash in water and examine. The organism is a rod-shaped diplococcus surrounded by a capsule (Simon).

BLOOD CHANGES.

The most common and characteristic blood phenomenon is leucocytosis. The normal number of leucocytes per cubic millimeter does not exceed ten thousand. In pneumonia it may increase to 30,000 or 40,000. Osler has seen 63,000. Leucocytosis increases with temperature and disappears with crisis. Its absence portends grave consequences. Its persistence means delayed resolution. Since leucocytosis does not occur in influenza, its presence is of diagnostic value.

THE URINE IN PNEUMONIA.

The quantity is diminished to one-third or one-half. Uric acid and urea are increased and large deposits of pigmented urates occur at critical stages. They are stained brown or red. The specific gravity is 1025 to 1035. Excess of mucus causes the fluid to become quickly alkaline. The chlorides are greatly diminished or even absent during the first and second stage, or even until convalescence sets in. Their return is a favorable indication. The sulphates increase. Nearly fifty per cent. of all the cases show albumin during consolidation and its presence is distinctly unfavorable. Diminution of the total solids is also unfavorable. Sometimes nephritis is intercurrent or is a sequel of pneumonia.

DIFFERENTIAL DIAGNOSIS.

Pneumonia in the first stage resembles pulmonary œdema. The latter differs by the presence of liquid crackling râles occurring at the most depending portions of the lungs and on both sides, while pneumonia is usually confined to one lung. Further, the frothy sputum, the general distribution of the râles, their coarser character, the cyanotic lips, the noisy breathing, the absence of fever, in œdema, prevent error. When not the immediate precursor of death, œdema is not an acute condition.

In the consolidation stage it may be confounded with pleural effusion. It differs from the latter in that bulging is absent, vocal fremitus instead of being absent is increased, by the fact that the percussion note is never wooden or absolutely flat and that its area does not move with changes in the position of the patient, that the respiration sounds are intensified and tubular instead of absent in pneumonia, and that when occasionally present in effusion they are indistinct, distant or deep-seated. Pleurisy is often without râles

and the sputum is frothy. Pneumonia does not displace any of the organs.

From phthisis it differs both in the history and in the course and progress of the disease. Tuberculosis advances from the apex downward. Pneumonia is confined oftenest to one lobe and that the lower lobe, is much more acute, the hectic flush is absent as are the night sweats. The character of the cough and sputum is different, there are no tubercle bacilli present. The pulse-respiration ratio is inverted, that is to say, in pneumonia the pulse is slower than called for by the respiration rate, while in phthisis it is faster. The dyspnœa of pneumonia is much more striking than that of phthisis. It is only in those cases of acute phthisis that appear after pneumonia, that the physician must be on his guard.

BRONCHO-PNEUMONIA.

Synonyms: Capillary Bronchitis, Lobular Pneumonia, sometimes Catarrhal Pneumonia.

Definition. An inflammation of the terminal bronchus and the vesicles which constitute a pulmonary lobule, due to the invasion of the lung by various kinds of microbes. It is lobular, in contradistinction to lobar, pneumonia.

Holt's statistics give a proportion of two cases out of every three as secondary to some other disease.

Bacteriology. The organisms most commonly found in broncho-pneumonia are bacillus influenza, micrococcus lanceolatus, streptococcus pyogenes, staphylococcus pyogenes aureus et albus, Friedländer's bacillus pneumoniæ, also the organisms of the associated disease when the process is secondary, as the bacillus of diphtheria. The infection is almost always mixed.

In typical form broncho-pneumonia is a disease of childhood, usually attacking children under two years. It is either a primary disease or is secondary to bronchitis, measles, whooping cough, diphtheria, ileo-colitis, scarlet fever or influenza, in the order named (Holt). It also follows other diseases but less frequently. Wilkes mentions it as occurring after extensive burns of the skin. It occurs in the course of typhoid fever. It follows the inhalation of foreign substances, giving a distinct type, the so-called inhalation or deglutition type, due to benumbing of the larynx, as after apoplexy, in uræmia, and post-operative pneumonia.

Rickets and malnutrition are predisposing causes and furnish a fruitful mortality. In the aged it often terminates a chronic bronchitis.

The disease is bi-lateral but one side usually bears the brunt of the attack. The lesions are small areas of consolidation, often surrounded by areas of over-distended cells alternating with small areas of collapsed lobules, which are depressed below the surface. The consolidated spheres change from brown to gray, and then to yellow with age. They may be separate and remain fairly scattered, but some aggregate and by their confluence form large racemose bunches of consolidation, with interspersed air cells, so that an entire lobe is never solidified. The lower lobes are affected oftenest and collapsed lobules are found near the base of the lungs. The small bronchi, the alveoli, and occasionaly even the large bronchi are filled with inflammatory products and proliferated cells. When pressed they exude purulent mucus.

The cases terminate by softening, abscess, gangrene (bronchiectasis often so terminates), return to normal or become chronic (chronic pneumonia). So much of the morbid anatomy is absolutely necessary for a proper understanding of the various signs and symptoms encountered.

Associated Lesions. The bronchial glands enlarge but do not undergo softening. The right heart dilates, due to the obstruction encountered in the lungs, and this leads to venous engorgement of the organs connected with the systemic circulation, as the spleen, liver, and kidneys. Intestinal catarrh is for this reason often associated.

Course and Symptoms. The primary attack often sets in with a convulsion, sometimes with a chill, the temperature rises rapidly to 103° and varies between 103° and 105°, but is fairly constant. Cerebral symptoms may mask the pulmonary lesion, or what is more likely, the disease may be mistaken for lobar pneumonia.

The secondary form begins as does the primary with accession of fever, cough and dyspnœa, rarely with chill. The fever is more irregular and ranges between 102° and 104°. Morning temperature may be higher than evening. The cerebral symptoms if they occur, come on early and go quickly. Breathing is from fifty or sixty to eighty per minute. (Compare lobar pneumonia.)

Physical Signs. Inspection. Patient rests propped up in bed with the head high, respirations are short and shallow, alæ nasi are

widely dilated. The face, at first pale, soon becomes livid and then cyanotic. The expression is anxious. The breathing is labored, the inspirations are short, the expirations are prolonged, there is little true expansion. The sternum rises, but there is recession of the lower interspaces, the submammary and epigastric regions, with inspiration. The tongue is dry, the skin hot and dry or alternates with perspiration. The pulse is small, quick, rapid and may be uncountable. The dyspnœa is painful, but the efforts to breathe subside somewhat under the influence of the non-oxygenated blood. In cases which survive dyspnœa gradually fades. Emaciation is rapid.

Palpation. Fremitus increases with the amount of consolidation.

Percussion. The note is normal as long as the areas affected are few and scattered. As coalescence occurs the resonance becomes impaired. This usually develops after forty-eight hours. The association of coalescence and collapse gives flatness, but in the most common form of the disease the patches are scattered and much of the lobe remains crepitant. Early in the disease the note may be hyper-resonant in front and along the over-distended edges.

Auscultation. Breath sounds are harsh in the upper lobes, feeble, absent or bronchial at the base, depending on the relative amount of the three factors which influence them, viz: consolidation, collapse and hyper-distention. As the bronchioles fill with exudate, blowing breathing predominates and expiration may be jerky and grunting. The râles, at first fine and subcrepitant, soon become higher and whistling and the fine crackling râles are not limited to inspiration.

If bronchiolitis occurs râles become metallic and high-pitched, associated with bronchial breathing and bronchophony. Rhonchi, sibilant râles and other signs of bronchitis vary with the extent to which the tubes are involved. Vocal resonance increases with consolidation.

Collapse is indicated by retraction of the side, absent breathing sounds, absent vocal resonance and fremitus. The percussion note of collapse is not as dull as that of consolidation.

The cough is hard, distressing and even painful. Occurring secondarily to bronchitis, the free secretion ceases. A cough which was loose and painless now becomes short, frequent, dry and painful. The secretion becomes viscid and is coughed up with difficulty. It may be blood-tinged but is never *rust-colored* as in pneumonia. In children expectoration is absent.

Diagnosis. The secondary forms are easiest diagnosed. If, during convalescence from one of the diseases mentioned, a child has an accession of fever with cough, a rapid pulse, decided increase in the respiration rate and if, on auscultation fine crackling râles scattered about the base of the lungs are heard, one may safely expect broncho-pneumonia to intervene.

The disease closely resembles pneumonia if the lobules coalesce into large masses. Broncho-pneumonia occurs oftenest before the second year, while lobar pneumonia is rare before the third year. Pneumonia is unilateral; broncho-pneumonia is bilateral. The first is primary, the second is oftenest secondary. Pneumonia shows fewer remissions in temperature and the crisis occurs about the eighth day.

From tuberculosis the diagnosis is often made by postponement. It is an aid to know that acute miliary tuberculosis, without softening, is more common in children of this age than the caseous form, but both occur. In tuberculosis the upper lobes suffer oftenest, in the pneumonias, the lower ones, and typical signs may be detected in the *vomited* matter, shreds and tubercle bacilli may be found. From meningitis it is differentiated by recalling that brain symptoms accompany the onset of many diseases of childhood, and the dyspnœa and the cough soon draw attention to the lungs.

EMBOLISM OF THE PULMONARY ARTERY.

Sometimes called Embolic Pneumonia, Hæmorrhagic Infarct or Pulmonary Apoplexy.

Definition. A conical extravasation of blood into the lung substance resulting from the lodgment of an embolus in a branch of the pulmonary artery. The embolus must arise from a thrombus which has formed somewhere in the venous system, and which, becoming detached and passing through the cavities of the right heart, ultimately blockades a branch of the pulmonary artery. During pregnancy such a thrombus may form in the venous system of the pelvis or in the femoral vein (phlegmasia alba dolens). During typhoid fever it may arise from thrombus of the saphenous or femoral vein. So also in the course of septic diseases, of pleurisy or phthisis, thrombi are apt to form in the veins mentioned or in the jugular vein. In middle ear disease they may form in the lateral sinus. Further, emboli may result from clots formed within the heart itself, in dis-

eases of that organ. Fat emboli are sometimes found after death from diabetes.

Symptoms. A small embolus may cause only a transitory œdema or hyperæmia which soon passes away.

Septic emboli cause inflammation, and gray hepatization follows the wedge-shaped infarct. This may be succeeded by abscess or gangrene. The typical lesion following pulmonary embolus is infarct, which is a form of pulmonary hæmorrhage. The area is cone-shaped, the base reaching the surface, the apex corresponding to the point of obstruction. If small, they give rise to few symptoms. If, however, a large area is involved, the attack comes on in a moment, the patient is seized with agonizing pain and the most intense dsypnœa, the heart is irregular and tumultuous, or weak and imperceptible, and death may result within a few minutes.

In severe but not fatal cases the symptoms are somewhat as follows:

Inspection. Patient is anxious, breathing is rapid and panting, but is *unobstructed.* Sometimes the Cheyne-Stokes type supervenes, lips are blue, skin cold and moist. Heart becomes gradually quiet and feeble.

Palpation shows increased fremitus.

Percussion reveals impaired resonance over the limits of the infarct.

Auscultation shows harsh inspiration, somewhat lengthened expiration, crepitant and subcrepitant râles, and even bronchial breathing.

Blood appears in the expectoration after twenty-four to thirty-six hours. In some cases involving large areas the signs are those of croupous pneumonia, but the outline of the lobe is not followed: the disease is unaccompanied by pyrexia, and comes on much more rapidly than is possible with pneumonia.

Occurring in the course of mitral disease the symptoms are: pain in the side, dyspnœa and possibly hæmoptysis, with the expectoration of dark, blood-stained sputum. A local pleural friction sound may mark the site of the infarct. Deep percussion may reveal tenderness (Head).

Diagnosis. The suddenness of the onset, the character of the dyspnœa—the patient breathing deeply, yet conscious of a lack of

air—the agony, the absence of signs of obstruction, should suggest the nature of the disease.

Asthma is accompanied by sonorous and sibilant rhonchi.

Laryngeal obstruction shows signs of impeded air entrance.

Œdema shows fine crackling râles over the bases of both lungs and is painless.

Infarct is unilateral.

The knowledge of a coexisting phlebitis, or of heart disease, aids materially the diagnosis. If seen after the onset and without a knowledge of the history of the case, the disease may be mistaken for pneumonia. Non-septic cases are afebrile. Septic cases are followed by chill, rigor, hectic, and the familiar chain of septic symptoms.

PULMONARY ŒDEMA.

Definition. An effusion of serous fluid into the air vesicles and interstitial tissues of the lung substance. It occurs in the lungs, as elsewhere, by reason of disturbances of the circulation and changes in the vessel walls, hence is associated to a more or less extent with all diseases producing pulmonary congestion. It gives rise to few symptoms except an aggravation of the existing symptoms of the associated disease.

As an independent entity we consider the pulmonary œdema which frequently terminates a variety of acute or chronic diseases, and is therefor spoken of as terminal œdema. Such diseases are acute and chronic nephritis, cardiac diseases, various anæmias and diseases of nutrition and cerebral diseases. It is prone to occur in maladies in which general dropsy exists. Acute œdema may follow the embolic obstruction of the pulmonary artery just described. Sometimes it follows the rapid removal of large quantities of fluid from the pleural cavity by thoracentesis. The transudate may be watery serum or blood-stained. Very acute œdemas are almost entirely serous.

Except in the case of the rapidly produced œdema just mentioned, the symptoms come on slowly. They are an increasing sense of suffocation, increasing difficulty of breathing, some cyanosis, a weak pulse and a cough which is short and dry. The œdema here begins at the base of the lungs and mounts, the symptoms increasing therewith. If suddenly developed, the above symptoms are much aggravated, the dyspnœa is severe and there is expectoration of abundant frothy sputa, which may be blood-stained or hæmorrhagic. The

dyspnœa depends on the number of air cells occupied. The malady is afebrile, bilateral.

Percussion gives areas of impaired resonance, amounting to dullness over the bases and dependent portions of the lungs. Large effusions give marked dullness.

Auscultation. The vesicular murmur is feeble or absent. Râles are fine or coarse, moist or liquid, according to the extent of the involvement. In extreme cases the breathing is bronchial, and coarse, rattling râles are audible at a distance.

Broncho-pneumonia intervening as a secondary disease is the only affection likely to be confounded with pulmonary œdema, but the mode of onset, the age, the fever, the glairy, tenacious expectoration and the scattered areas of dullness in the latter will clear up the doubt.

SECTION VIII.

PULMONARY TUBERCULOSIS OR CONSUMPTION.

Definition. An acute or chronic febrile infective disease of the lungs, caused by the bacillus tuberculosis, characterized by diffused tuberculous infiltration, the formation of tubercles which undergo caseation or sclerosis and which may ulcerate or calcify.

VARIETIES.

A. Acute phthisis. 1. Tuberculo-pneumonic phthisis (Osler). 2. Acute tuberculous broncho-pneumonia.

B. Chronic ulcerative phthisis.

C. Fibroid phthisis.

D. Miliary tuberculosis of the lungs.

The first form, acute pneumonic tuberculosis of the lungs, known also by the name of "galloping" consumption, phthisis florida, is characterized both by its diffuseness and its rapidity. It occurs in both children and adults. Many cases occurring in children are mistaken for simple lobar-pneumonia. Since an entire lobe or even an entire lung is affected, and inasmuch as physical signs are identical with those of pneumonic hepatization, in which condition the lung is found to be post-mortem, the mistake is excusable. The upper, then the lower lobe, then the entire lung, is the order of frequency of involvement.

The physical signs are those of consolidation; increased frequency of respiration, dyspnœa, percussion dullness, marked increase in fremitus, feeble, vesicular murmur followed by suppression and then bronchial breathing. This tubular breathing becomes very intense and is marked as early as the fourth day. The characteristic crisis does not occur by the tenth day and the sputum becomes muco-purulent and changes to green color (Traube). Tubercle bacilli are now found in the sputum, which settles the diagnosis.

In the second form, which Osler denominates acute tuberculous broncho-pneumonia, which form he states constitutes the majority of cases of phthisis florida, especially in children, the lesion is a blocking-up of the small tubes with cheesy matter, while the air cells of the lobule are filled with the products of catarrhal pneumonia. These areas are separated by areas of crepitant tissue, but by their fusion an entire lobe may be rendered nearly solid. Owing to the interspersed islets of crepitant tissue the physical signs are indefinite. The first positive signs are usually those of deposits at one or the other apex.

The respirations are markedly increased in number, the subclavicular expansion is diminished. Areas of diminished resonance are found, more generally at the apices. Over these areas the vocal resonance and fremitus are increased, percussion sounds impaired, pitch raised, and the approach to flatness is directly in proportion to the volume and density of the involved tracts. Auscultation shows the absent vesicular sounds, replaced by rude or harsh tubular breathing, associated with râles, numerous and varied in character. The cough, hacking, troublesome and almost continuous, at first dry, soon is accompanied by expectoration which very early shows the presence of tubercle bacilli and elastic fibers. The tongue is dry and brown, the fever high. A few of these cases survive the acute stage only to become chronic. This form is especially prone to follow measles and whooping cough.

Chronic Phthisis. Ulcerative Tuberculosis of the Lungs. As first pointed out by Laennec, and more recently in the admirable analysis of J. Kingston Fowler, 1888, the primary lesion is most often situated from an inch to an inch-and-a-half below one or the other apex, nearer the external and posterior than to the anterior surface, whence it spreads downward. As has been pointed out in the section on topography, the lung projects from one to one-and-a-half inches above the clavicle, hence this point of infection would lie anteriorly under the center of that bone. The situation of the focus towards the posterior surface accounts for the occasional presence of demonstrable dullness in the supra-spinous fossa, when it cannot be made out anteriorly. The right apex is the seat of the primary lesion more frequently than the left. Fowler further calls attention to the fact that the lower lobe is usually affected when the physical signs of disease at the apex are sufficiently definite to allow of the diagnosis of

phthisis being made. The point of involvement is again from an inch to an inch-and-a-half below its apex, which corresponds on the chest wall to a point opposite the spine of the fifth dorsal vertebra. Have the patient reach the arm of the affected side around his opposite shoulder and place his fingers upon the spine of that scapula. The point in question is marked by the inner edge of the abducted scapula and the spine named. Cavities are usually first formed where the lesions are oldest, and must be as large as walnuts before they may be positively diagnosed by the signs. Hence patient and repeated examinations of the points indicated should be made.

The next most common seat of cavity after the localities described is in the second or third interspace, anteriorly, outside the mid-clavicular line.

It is of extreme importance in examining a suspected case of phthisis that the orderly sequence laid down in the preliminary chapter be followed carefully, and it may be said to the credit of the diagnostician that in the vast majority of the cases a correct conclusion may be arrived at by the intelligent interpretation of the physical signs, before it may be reached by a bacteriological examination, and with no less certainty. Before attempting to explain the physical signs which accompany the tubercuIous invasion of the lungs in the chronic variety of the disease, it is necessary to name the lesions produced. These are:

a. Scattered and disseminated tubercles. These so long as they are few and separated give rise to no positive signs.

b. Caseous tubercular masses due to the union and breaking down of neighboring tubercular particles. The localities in which these masses are prone to occur earliest have been pointed out in this section. The signs to which they give rise vary with their situation and extent, and are considered below.

c. Tuberculous cavities (situation noted). These may have soft walls undergoing ulceration and extending their areas by breaking down, or the walls may be composed of the so-called pyogenic membrane, in which case extension is slower. The formation of the cavity wall, the size, the situation (superficial or deep), the nature of the opening into it, whether full or empty, all modify materially the signs.

d. Cavities formed by dilatation of the bronchi, called bronchiectasis (q. v.). The form of such cavities may be cylindrical (small bronchi) or globular (large bronchi).

e. The various forms of pleurisy, some of which are invariable accompanists of phthisis during some part of its course. Three common variations are worth naming. 1. Adhesive, over the infiltration area, which results in a local glueing together of the two layers. 2. Perforative, into the pleural sac, resulting in empyema and pneumothorax. 3. The entire pleura may become the seat of tubercular infiltration, be studded with tubercles, thickened and may cement the lobes into a single mass.

f. Pulmonary concretions. These may not be differentiated by the physical signs, but are often coughed up, as are also bronchial concretions found in chronic bronchitis with dilatations. Those of phthisis differ from those of bronchitis by being more irregular, non-uniform masses instead of pebble-like.

The physical signs are best considered under three stages:

The *incipient stage*; the *stage of complete consolidation*; the *stage of softening and formation of cavities.*

THE INCIPIENT STAGE.

Pulmonary tuberculosis is met with in chests of all shapes and capacities but the long, narrow chest is the prevailing type. Here the intercostal spaces are wide, the costal angle narrowed and the vertical direction of the ribs increased. The scapulæ project like wings, often one more than the other. In another type the antero-posterior diameter of the chest is lessened, the sternum is sunken, the subclavian spaces flat and the clavicles prominent.

Inspection may be negative. A few scattered tubercles give rise to no phenomena appreciable by our coarse senses, but as soon as they are sufficient in number to interfere with the elasticity or to increase the conductility of the lung tissue, they produce objective signs. On the other hand, if any wasting has occurred the clavicles are rendered more prominent and there may be slight flattening below one or the other collar bone. Expansion may be slightly diminished in the same region. Deficient apical expansion is an early and important sign. It may best be estimated by taking a position behind the patient, who should be seated, and looking downward over the shoulders. The breathing will be somewhat accelerated but shallower than in health, and the apex beat may show acceleration with marked quickening of the impact.

The wavelike shadow caused by the rhythmical rise and fall of the

diaphragm, known as Litten's sign, which is more fully described in the section upon adherent pericarditis, is seen to be altered early in pulmonary tuberculosis. The change, for the most part, consists in a limiting or shortening of the normal shadow on the affected side. The sign is important and always should be searched for in suspected cases.

Palpation. Deficient expansion may best be gauged by standing behind the patient and alternately placing the fingers in the subclavicular spaces and the lateral regions of the chest and noting the relative motility. So also, by placing the thumbs in the supraclavicular spaces, while the fingers are placed in the upper intercostal spaces, one may estimate the relative expansion of the apices with fair accuracy. (See Fig. 17.) Vocal fremitus is increased wherever the condensation is of sufficient degree to assist sound conduction In the early stages it is difficult to estimate slight increase in conduction, especially when it is recalled that deposits are more apt to occur on the right side, and that normal fremitus is somewhat greater on this side. If, however, fremitus is greater *on the left side* than on the right, the fact is significant. Pleuritic thickening causes diminution instead of increase of the touch fremitus.

Mensuration. The tape may show deficient expansion on one side, or, what is more general, the tape encircling the body immediately under the arm-pits does not show the amount of expansion which is to be expected. This obtains early in the disease.

Percussion. Changes may be recognized on percussion which are not made manifest by palpation. The first positive sign of infiltration is dullness and its most usual seat is immediately underlying the clavicle. It is most surely discovered by direct percussion on that bone, without intermediation of the pleximeter, although it may require a cultivated ear to detect the change. Its note is higher and shorter than normal, and is the note of impaired resonance. The difference in the two sides may be marked, or both may be affected. The impairment when not at the apex, is oftenest just below the clavicle. The note should be elicited during natural breathing, on forced expiration, on forced inspiration and with the mouth held open, as directed by Flint. A comparison made between corresponding areas while the breath is held after forced inspiration, most surely detects defective resonance, when present.

Auscultation. When positive percussion changes can be elicited

abnormalities on auscultation are invariably present, and in some cases even precede impaired resonance. The earliest auscultatory sign is, generally speaking, feeble breathing, owing to a lessening of the tidal air entering the vesicles of the affected area. Make careful comparison between the two sides during quiet breathing. Inspiration on the diseased side may be inaudible. Next in order of sequence is a prolongation of the expiratory murmur. About the same time, or a little later, the inspiratory sound grows harsher. The type of breathing now becomes broncho-vesicular. Vocal resonance is increased in direct proportion to the amount of consolidation, yet in not a few cases it will be found absent by reason of early pleurisy having thickened the intervening membranes, thereby masking its effect, as noted under touch fremitus. Bronchitis, when present, is manifested by râles. A soft bruit heard in the pulmonary or subclavian artery is often present, and Da Costa states: "A murmur is, indeed, at times present in the pulmonary artery long before any other physical indication of tubercle is discernible." Also interesting is the so-called cardio-respiratory murmur met with in phthisis, caused by the propulsion of air from the tubes by the impulse of the heart. It is best heard during inspiration, in the antero-lateral regions of the chest. In time the murmur is systolic, and is described as a whiffling bruit. (See heart murmurs.) To sum up the signs already elaborated, which allow of the diagnosis of beginning tuberculosis with almost as great certainty as does the finding of tubercle bacilli in the sputum, and often long before their occurrence therein, we append the following:

Signs of incipient phthisis.

- Modification of vesicular murmur at apex.
- Adventitious sounds, arterial or respiratory, limited to the apex.
- Percussion dullness in clavicular area or supra-spinous fossa.
- Increased fremitus.
- Impaired expansion or flattening of anterior thoracic surface.
- Cough.
- Shortness of breath.
- Sustained rise of temperature with probable morning maxima.
- Modification of Litten's diaphragm sign.
- Failing health.

STAGE OF CONSOLIDATION.

The second stage gives rise to signs more positive and more pronounced than the first.

Inspection plainly shows loss of flesh, prominence of clavicles, ribs and interspaces. A hectic flush may be noted. Flattening is

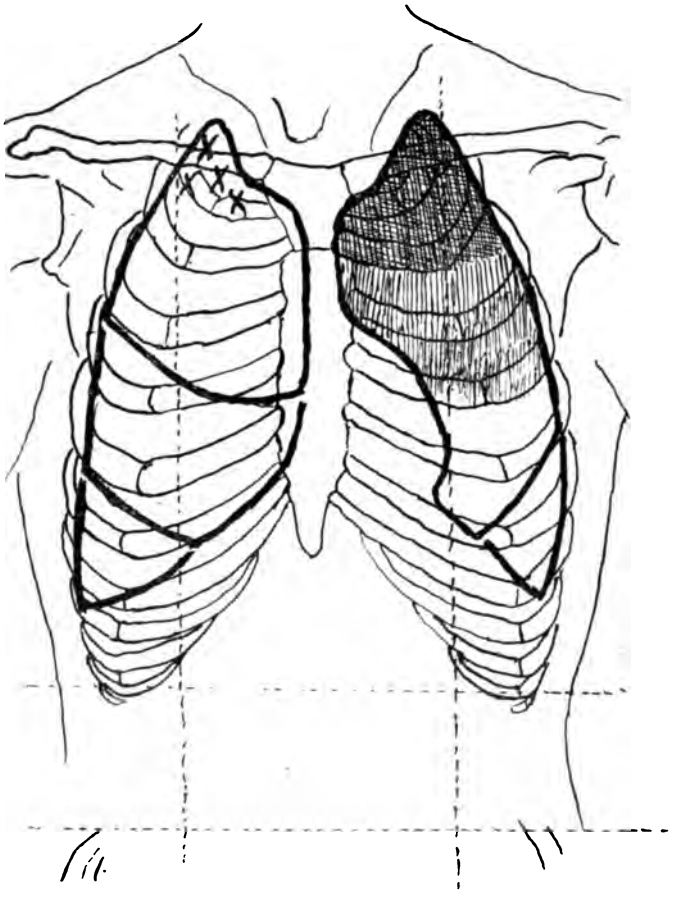


Fig. 37—Extension of tuberculous lesion (Patton). Dark shade—primary lesion; light shade—extension of primary lesion; X—secondary lesion of opposite apex.

most apparent anteriorly and the breathing excursion is lessened both with quiet breathing and during forced inspiration, when lack of expansion is striking. Expansile efforts may be painful.

Palpation shows increased fremitus except, as noted in first stage, where pleurisy has resulted in thickening. The skin is hot and dry.

Percussion shows positive dullness over the affected areas, which contrasts markedly with the unaffected parts where the respiration is augmented and the note preternaturally clear. Both apices may be

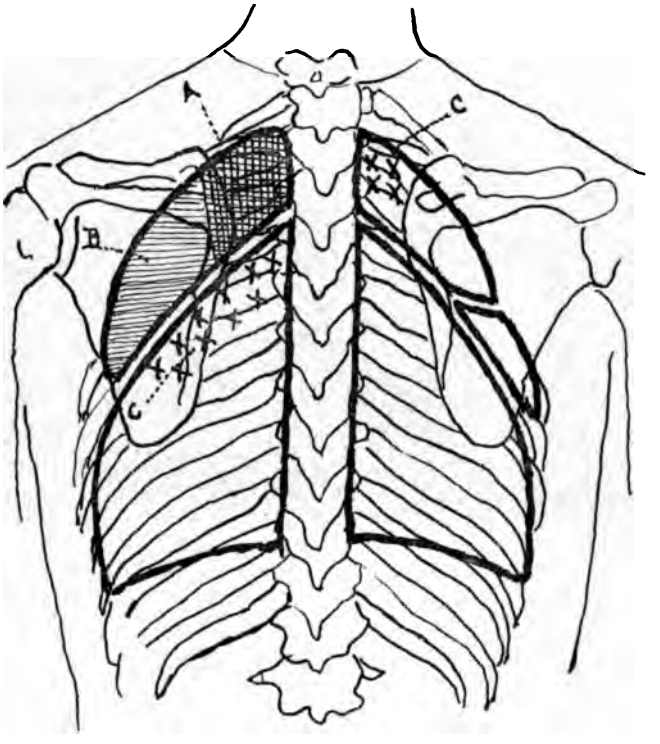


Fig. 38—Showing relation of lobes to posterior chest wall, also extension of tuberculous lesion (Patton). A Primary lesion. B Extension of primary lesion. CC Secondary deposits.

affected. The plexor finger notes increased resistance. Pain on percussion is often complained of. Hard, board-like tympany is not encountered in recent cases but is found in old chronic cases with extensive fibroid changes. Caseous consolidation, as noted under pneumonic phthisis, has a tubular or bronchial quality. Percussion should be practised in the supraspinous fossæ and in the intrascapular regions, for reasons already given. In muscular and fleshy people not much information may be gained thereby, but in thin, and especially

in emaciated individuals, the returns are of great value. Myoidema is the name given to the local contraction and bulging of the muscles, when subject to irritation, such as occurs in direct percussion of the pectorals. It is encountered in thin, nervous individuals, and, although often seen in phthisis, is not of any special significance.

Auscultation. Vocal resonance is increased. The breath sounds are harsh.

The disappearance of the vesicular elements and the approach toward pure bronchial breathing are in direct proportion to the amount of consolidation. The inspiratory portion of the sound is shortened and the expiratory part is prolonged and blowing. When consolidation surrounds a bronchial tube, typical blowing respiration obtains. The signs are apparent in the supraspinous fossæ as well as in front. Bronchophony, a high degree of vocal resonance, may be present. It denotes consolidation. The presence of pleurisies may give rise to fine superficial friction sounds or cracklings, and extensive bronchitis may, by its noisy signs, interfere with auscultation.

Acquired lesions of the cardiac valves are certainly rarely encountered in the subjects of pulmonary tuberculosis, especially in the chronic forms of the disease; so rarely, indeed, that the late Henry Formad denied their existence.

As pointed out, however, under the caption Congenital Cardiac Defects, the subjects of congenital valvular lesions are very prone to acquire pulmonary tuberculosis.

THIRD STAGE. SOFTENING STAGE. CAVITY FORMATION.

During this stage a portion of the lung is undergoing softening while in other portions consolidation is proceeding by reason of continued infiltration. Hence the signs vary. The persistence of moist râles and cracklings in areas previously consolidated indicates softening.

Inspection. Hectic has increased. Alternate hot, dry skin and cold, clammy perspiration are noted. Emaciation has made marked encroachments. The limitations of movement during respiration are still more restricted and the flattening under the clavicles more pronounced than in the earlier stages. The superficial veins over the thorax attract attention by their prominence and fullness. Tyson draws attention to upward retraction of the heart in cases involving the left upper lobe. It must be rare. He also notes that the area of

cardiac impulse enlarges upwards and pulsation may be visible in the third and fourth left interspaces.

Palpation. Vocal fremitus is still increased in spite of cavity, since cavities form in the midst of consolidated areas and are surrounded by compact substance which increases conduction. Osler remarks, "In the later stages, when cavities form, the tactile fremitus is usually much exaggerated over them." But considerable thickening of the pleura diminishes this fremitus. If air cannot get into a cavity by reason of its bronchus being closed, the fremitus is not increased. Rhonchial fremitus may be present.

Percussion over cavities gives neither as positive nor as unvarying results as over consolidation. If the cavity wall be thick and the substance between it and the surface dense, the percussion note will be dull as in consolidation, though heavier percussion may elicit tympany. If dense, but thinner walls intervene, the sound is a mixture of dullness and tympany, difficult to describe. Very thin walls yield sounds which are truly tympanitic or metallic. Amphoric and cracked-pot sounds often may be elicited. To demonstrate the cracked-pot sound, percuss with a firm, quick stroke, while the patient with suspended respiration, holds the mouth open. If a cavity underlies a layer of healthy tissue, which would be a seemingly rare condition, the note is clear, especially on light percussion. Percussion sounds are much modified in the presence of cavities by the act being performed with the mouth alternately opened or closed. With the mouth closed, the pitch is lowered and the vibrations longer. This is known as Wintrich's changed note. Da Costa points out that over cavities the heart sounds are heard with extraordinary clearness and that there is often seen a "wavering impulse" in the second intercostal space.

Auscultation. The breathing sounds vary with the relative degree of consolidation and excavation. Over the hardened areas we still have bronchial sounds, and if these are mixed with small bubblings and cracklings, subcrepitant râles, it indicates liquefaction or beginning excavation. Cavities give rise to new sounds. It is worthy of note that numerous small, isolated cavities without much fibroid deposit or pleural thickening, may exist at the apex and yet the percussion note remain unaltered, although the auscultatory sounds will be greatly modified and out of harmony with the percussion findings. Cavernous breathing describes the tidal air entering and leaving an

excavation. When the walls of the cavity are firm and unyielding, the note of respiratory rhythm is higher pitched, metallic and echo-like. This is amphoric breathing. Large bubbling sounds occur when air passes into a cavity partially filled with liquid, termed gurgling râles. Cavities which give forth the amphoric respiratory sounds will, when they contain liquid, produce metallic tinkling.

The voice sounds as modified by cavities, are interesting and distinctive. Cavernous voice, like cavernous breathing, is the hollow, resonant quality imparted to the sound by reason of its traversing an excavation. Amphoric voice has added thereto an echoing, metallic or musical quality, and is higher in pitch than the cavernous voice. Pectoriloquy, both spoken and whispered, when present is a distinctive sign of cavity. The term has already been defined. In apical cavities the heart sounds are often distinctly heard and occasionally an intense systolic murmur is transmitted into the cavity. Over left apical cavities, gurgling sounds or crackling sounds, synchronous with the heart impulse, may be heard. They are caused by an impact communicated to a cavity partially filled with fluid. Walsh describes a case in which the succussion splash was obtainable, as described under pneumothorax.

AUXILIARY SIGNS OF PHTHISIS.

Fever. The deposition of tubercles is accompanied by elevation of temperature, hence fever is an early and characteristic sign. The maximum is between two and six P. M. Early fever is generally remittent. Later both intermittent and remittent types occur. Hectic, which is in reality septic fever, due to the absorption of tuberculous products, is a more or less regular rise and fall of temperature varying between wide ranges, rising to 102°—104° and falling to subnormal, even 96° being not unusual. This type prevails in the softening and cavity formation stage.

Cough. This symptom has already been touched upon. At first so slight that the patient will deny its existence, it often grows to be the most distressing and intolerable of the symptoms, causing the patient to lose sleep or, as is sometimes the case, exciting intractable vomiting, thus contributing to the rapid emaciation of the afflicted. Its early characteristic is hacking, unaccompanied by expectoration; technically, unproductive. Then a glairy mucus is coughed up. If the larynx is involved early the cough as well as the voice is husky.

Spells of coughing followed by copious expectoration denote cavities or bronchiectasis. Oftenest such paroxysms occur on waking. Some cases progress to solidification or even to cavities with very little cough.

Expectoration follows cough, although the cough may have remained dry for a long period. At first scanty, it increases rapidly and may be as much as 250 c. c. in twenty-four hours. Sometimes distinct apical manifestations or extensive consolidation are accompanied with insufficient expectoration to allow of an examination. The early expectoration is glairy, mucoid, containing sago-like grains, alveolar cells which have undergone myelin degeneration. (Thudichum.) Presently grayish or greenish masses are observed and are the first differential constituents of the sputum. They should be examined microscopically. After this the expectoration becomes more mucopurulent, may be blood-tinged. The nummular masses already described are of frequent occurrence, and are distinctive of cavity formation. Each mass is separate, greenish or gray-green in color, airless and sinks to the bottom of the cup when thrown into water. The odor of the sputum is sweetish, mawkish, unpleasant but not offensive. The sputum contains pus corpuscles, and epithelium from the entire tract traversed by the air—the mouth to the alveoli. The latter cells are most numerous. Blood discs, particles of food, oil drops, elastic tissue fibers and bacilli are other constituents.

Elastic fibers are derived from the blood vessels, the bronchi, the alveoli or from particles of ingested food. Rinsing the mouth before collecting the sputa will obviate a mistake as to food tissue. It is to be remembered that such tissue may be retained in the mouth for several days. Boiling the picked-out masses with liquor potassa or soda in a test tube allows the elastic tissue to fall to the bottom of the tube, when it may be examined microscopically. Osler states that a method quite as efficient is to spread the masses into a thin layer between two glass plates, which are then held against a black background. The elastic tissue shows against the background as opaque, grayish-yellow spots, which may be immediately examined by the low power, or by sliding the glasses apart may be picked out for examination on a slide. He states that milk globules and fragments of bread are similar in appearance, but that the eye soon learns to distinguish these. Fibers from the alveolar walls are branched or wreath-like interlacements and show the arrangement of the air-cells. Those from

the bronchi are elongated fibers, two or three together and form reticuli. Those from the artery may be sheet-like or fenestrated membrane, as though it were the intima of a vessel. Generally they resemble the fibers derived from the bronchi. Alveolar fibers indicate extensive erosion, softening and destruction. Epithelial cells from the alveoli are also found. These are large, oval or round non-nucleated cells about twice the size of a pus corpuscle.

The *tubercle bacilli* are the unfailing sign of tuberculosis. They are made apparent only by special staining methods, and microscopic examination. The simplest method is perhaps the following: Shake up commercial aniline oil in water until the solution is saturated. Filter out 100 c. c. of this solution. Next add fuchsin to absolute alcohol until the alcohol is saturated. Add 11 c. c. of the second solution to the 100 c. c. of the first, for the stain.

Pick out a small, cheesy-looking particle of sputum with a needle and spread on a cover-glass by rubbing against a second glass. Dry these slowly by holding a foot above a Bunsen burner (or alcohol flame). Cover the slide liberally with the staining fluid and hold near the flame until the fluid boils, after which it is washed in running water. It is now put in a 30 per cent. solution of nitric acid until decolorized, when it is again washed, mounted and examined.

The bacilli are colored red and appear as elongated, slightly-curved, sometimes beaded rods. In doubtful cases the covers are allowed to remain twenty-four hours in the stain. The number varies from a full field to one or two found only on repeated examination.

In disputed cases, where the symptoms are not confirmed by the microscopic findings, it is recommended to make cultures from the sputum. Directions therefor may be found in works on Bacteriology.

Hæmoptysis. By reason of the frequency with which it occurs and the serious results which attend it hæmoptysis has a specially important relationship to pulmonary tuberculosis. Blood may simply occasionally tinge the expectoration or it may be present in the sputa in considerable quantity. It occurs early and late in the disease and is more frequent in males than in females. It may be the first premonition of the invasion of the dread destroyer, or it may close the drama. The onset of the attack may be sudden or a premonition of its oncoming may be gathered from the staining of the sputa which often precedes it for two or three days. It may come on during quiescence, after exercise, after a fall or a blow upon the chest. The

idea that it follows sudden exertion, strain or excitement is not borne out by clinical experience. In many cases it comes on during sleep. In a proportion of the subjects both physical signs of the disease and bacilli are absent. In another proportion bacilli are found subsequent to hæmorrhage. In the third class, evidences of the disease are positive and bacilli found in the expectoration confirm the signs.

Hæmoptysis should, however, always excite a strong suspicion of tubercle and the cases subsequently should be carefully watched.

Hæmoptysis occurs in sixty or eighty per cent. of all cases of pulmonary tuberculosis. The amount varies from a teaspoonful to a pint, being smaller when it occurs early in the disease. Even large hæmorrhages are seldom immediately fatal. In character, blood from the lungs is frothy, mixed with mucus, bright-red in color, unless it has been retained in a cavity, when it is darker. After hæmorrhage the sputa may contain dark masses of blood or may be blood-streaked for several days. A careful examination of the hæmorrhagic mass often reveals small mucous nodules in which bacilli or elastic tissue may be found.

The general symptoms accompanying the loss of blood are noted under hæmoptysis. Some of the special symptoms are the great disturbance of mental balance and the febrile reaction, lasting from several days to two weeks, which succeeds every considerable hæmorrhage. This is attributed to the foci of broncho-pneumonia arising from the lung substance derived from the hæmorrhagic cavity which have been drawn into the pulmonary alveoli. The bleedings may be widely separated from each other or may recur at frequent intervals.

Dyspnœa is not increased in proportion to the acceleration of respiration, and is not marked even when respirations are very frequent. Dyspnœa may be cardiac by reason of enlargement of the right heart.

Perspiration. Drenching perspirations are a common symptom. These come on after cavity formation. They occur after the fever drop which takes place towards morning, hence the familiar term "night sweats". They may occur during the day. Sometimes they occur in the early stages and are the cause of the patient seeking the doctor.

The pulse is soft and compressible, though quick and frequent. Venous pulsation and capillary pulsation are often seen. With the progress of the disease the pulse weakens. Vomiting, excited by

coughing, is an unpleasant symptom coming on late in the disease. Pain either is due to coughing, when it is located at the base of the sternum, or it is due to pleurisy, when it is located over the lesion.

Diarrhœa is one of the late symptoms. When once established it is apt to prove intractable.

The clubbing of the fingers, as noted by Hippocrates, comes with the advance of the disease, but occurs also in chronic asthma, bronchitis, and more rarely in chronic cardiac diseases. It has been noted in cases of aneurysm. The finger-tips become bulbous and the nails curve over the finger tips. All of the fingers are not equally affected. (See Clubbing.)

The Urine in Tuberculosis. The quantity of urine voided is influenced by the diaphoresis, diarrhœa, pyrexia and even the quantity of expectoration. Thirst, which accompanies the febrile stage, increases the amount; the other conditions diminish it. It may be as little as 500 c. c. Uric acid and the sulphates change but little. Urea diminishes markedly during the daily febrile period, and increases in the same proportion during the sweating period. It often settles down as a pink sediment. Diarrhœa diminishes the urates here as in other diarrhœal diseases. The amount of chlorides varies with the quantity of food ingested and the incidental complications.

Ehrlich's diazo-reaction is sometimes obtainable. Its presence may be regarded as unfavorable. It is apt to be found in cases which are rapidly-progressing, and chronic cases which are nearing the end. A trace of albumin is frequent, but casts, blood and epithelium are exceptional. When present they indicate amyloid changes. If pus occurs in the urine it should be examined for bacilli.

The method of obtaining the diazo-reaction is described in the section on Urine.

State of the Blood in Pulmonary Tuberculosis. Cabot* makes the following statements:

1. The red corpuscles are usually normal, but the hæmoglobin is diminished. In some cases both are diminished.

2. The leucocytes do not change in character.

3. In the early stages of the disease the white corpuscles are normal, after an attack of hæmoptysis they show an increase.

**Year-Book of Treatment*, 1897.

4. If cavities are present there is no leucocytosis. If the leucocytes are increased, there are no cavities.

5. Pneumonic tuberculosis (extensive infiltration) may show marked leucocytosis, not invariably.

6. Fibroid tuberculosis shows no leucocytosis.

7. Pyrexia, due to the presence of pyogenic organisms, shows leucocyte increase. If not, there is no leucocytosis. If fever is absent there is no leucocytosis.

In contradiction to the fourth statement, which would be most important if confirmed, Stein and Erbmann state that one of the conditions of leucocytosis in pulmonary tuberculosis is the presence of cavities in the lungs, hence at present no definite conclusions can be reached on this point.

SECTION IX.

THE HEART.

Physiology. The heart is a double-cylinder muscular pump. Between the two cylinders no communication exists after birth. One side serves for the low service or pulmonary circulation, the other for the high service or systemic circulation. Each pump consists of two chambers, the receiving chamber or auricle, and the pumping chamber or ventricle. As soon as the auricles are filled they contract, forcing the blood into the ventricles, the contraction of which immediately follows. After the ventricular contraction a period of rest succeeds.

The contraction of the cavities of the heart is called the cardiac *systole*, the period of rest is called the *diastole*, the two periods make up the cardiac *cycle*.

The systole of the corresponding cavities of the two sides of the heart is exactly synchronous, that is to say, the two auricles contract simultaneously; and the simultaneous contraction of the ventricles immediately follows that of the auricles. While the auricles are contracting the ventricles are in a state of relaxation, and the relaxation of the auricles commences directly after the ventricular contraction begins.

The rate of the heart beat depends upon the duration of the diastolic pause, which lessens proportionally as the heart beats more rapidly.

Anatomic Relations. Encased in the pericardial sac, suspended apex downward from the great vessels, the heart largely occupies the middle mediastinum or interpleural space. It hangs obliquely behind the lower two-thirds of the sternum, projecting slightly to the right and considerably to the left of that bone.

That the heart is suspended by its vessels and not supported by the diaphragm is proved by the fact that the diaphragm separates from the heart during deep inspiration, as shown by the Röntgen rays.

Furthermore, the rays show that the heart chambers are not en-

tirely emptied at each systole, as was formerly assumed by physiologists (Beck).

Behind, its base corresponds to the sixth, seventh and eighth thoracic vertebræ, from which it is separated by the œsophagus and the aorta. In front, the base corresponds to a line drawn across the sternum at the lower border of the second costal cartilage, extending one-half inch to the right and one inch to the left of the sternum. Its lower border, *made up of the right ventricle*, is nearly horizontal. It rests lightly on the central tendon of the diaphragm, which separates it from the convex surface of the liver. The apex, which possesses a certain freedom of motion, strikes the chest wall at a point between the cartilages of the fifth and sixth ribs, at a point two inches below, one inch to the sternal side of the nipple, which point is 3¼ to 3½ inches from the midsternum in the fifth interspace. The right ventricle is anterior and lies directly under the sternum. Its lower *segment* occupies the fifth interspace, its lower border is on a level with the sixth cartilage. The organ projects slightly to the right, but considerably to the left of the breast bone.

The left ventricle lies chiefly behind the right, but its left border, which includes the apex, comes to the front in systole. This border lies wholly within the nipple line.

Perhaps DaCosta was the first to say that the heart of an individual is about the size of his fist. Roughly speaking, the comparison is fairly correct and useful. Cunningham gives the average weight of the heart in adult males as eleven ounces (310 grams), in females nine ounces (250 grams).

The Valves of the Heart. Each side of the heart is equipped with two valves. One guards the opening between the two chambers, the auricle and its corresponding ventricle, the other, placed within the ventricle, closes the aperture of the great vessel which springs therefrom, viz:—the aorta on the left and the pulmonary artery on the right side. Three of these valves are made up of three cusps, or leaflets, each, while the remaining one consists of only two cusps. This latter, named the *mitral* valve, closes the orifice between the left ventricle and its auricle. To the free edges of its leaflets are attached the *chordæ tendineæ*.

The corresponding opening on the right side, that between the right ventricle and its auricle, is closed by the *tricuspid valve*, com-

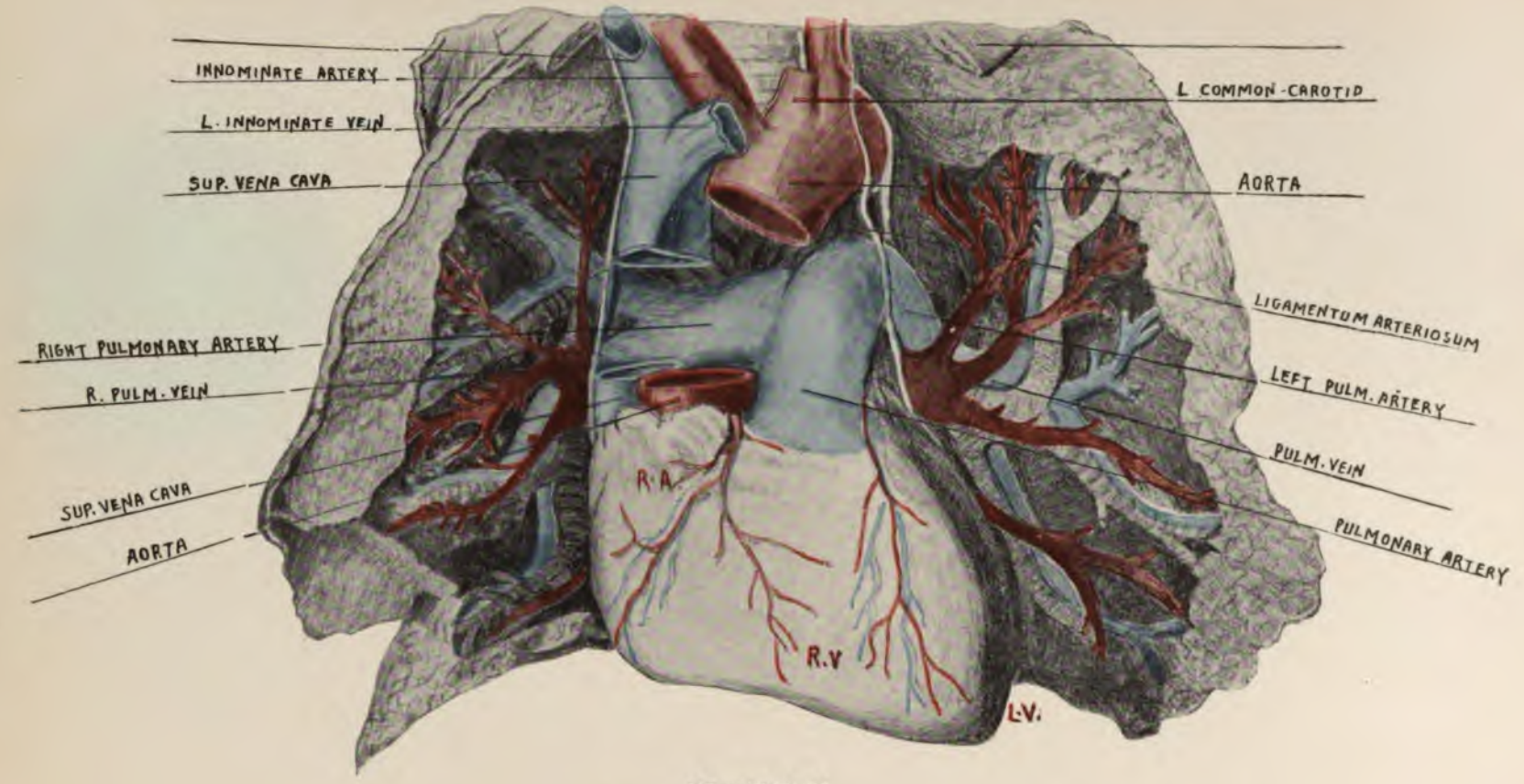


PLATE V.

POSITION OF HEART AND ITS RELATIONS.

Modified from Cunningham's Anatomy,
by permission of Wm. Wood & Co.

posed of three leaflets as its name indicates. Its movements are also limited by the chordæ tendineæ.

The aortic and the pulmonary valves close each their respective openings. Each is composed of three cusps or concave pockets whose edges are free. They are named respectively, the *aortic semilunar* and the *pulmonary semilunar valves.*

The Præcordium. That portion of the thoracic wall which covers the heart is called the præcordial region. It is roughly quadrilateral, and its boundaries are a vertical line drawn ¾ths of an inch to the right of the sternum, another parallel thereto passing just outside the apex, two horizontal lines, the first drawn through the junction of the manubrium with the gladiolus of the sternum (second rib), the second passing through the ensiform cartilage.

THE EXAMINATION OF THE HEART.

The method adopted for the examination of the lungs applies equally to the heart, and the same order should be observed, namely, the history of the case should be followed by inspection, palpation, mensuration, percussion and auscultation.

History. Much diagnostic aid is derived from the history of the case, since many symptoms point unmistakably to cardiac lesions. Especially the date of previous diseases, which may have stood in a causative relation, should be fixed and their nature carefully inquired into.

When the condition of the patient admits the standing position is the most advantageous for the examiner, who may himself either stand or be seated on a rather high chair. He thus avoids the humming noise arising from his own circulation, which is a fruitful source of annoyance to the examiner when he is obliged to assume a stooping posture.

Even bearing in mind the possibility of being accused of prolixity we urge upon the student the desirability of taking advantage of every reasonable opportunity to examine the normal heart with all the completeness of detail which would be used were the organ diseased. While percussion and auscultation are relatively the most important steps, the others are by no means to be neglected.

Inspection. We begin our inspection by noting the contour of the chest wall. In health the two sides are symmetrical or nearly so, but this symmetry may be gravely altered by disease. Occasionally we may detect a slight prominence or even protrusion over the seat of

the heart in perfectly healthy persons, especially those who practise habitually great physical exertion. Hypertrophy, or the accumulation of fluid within the pericardium, markedly emphasizes this prominence. Pericardial inflammation may leave as a sequence a very evident præcordial depression. We note the condition of the circulation, as manifested by the capillaries, the presence or absence of œdema about the eyes or ankles as evinced by pitting, the presence and character of the cough, the number and character of the respirations and the amount of dyspnœa. One often notes a peculiar anxious expression of the countenance accompanying certain heart lesions, which is most impressive, and not without significance.

The most important particular of inspection is the location of the apex. It is usually possible to recognize the apex beat by inspection, except in stout persons or in cases where the organ is retracted from the chest wall. Sometimes its absence is accounted for by its striking against a rib, instead of in an interspace. The impact is less apparent in women than in men, even when the mamma is retracted. It is more pronounced in spare individuals and in cases in which the organ is hypertrophied.

The location of the impulse has been given already as lying between the fifth and sixth ribs at an average distance of three inches to the left of the mesial line. In narrow-chested individuals it may be lower and in children it is often an interspace higher. In case the impulse is invisible it may be brought into view by causing the patient to walk briskly a score of paces, by bending him forward, or, if recumbent, by turning him well towards his left front.

Inspection should note not only the position of the apex beat, but its area, whether it be diffused or concentrated and the regularity of the succession. In health the area should not exceed one square inch. The location of the impact changes only slightly in the different positions assumed by the patient, but is less pronounced when the person is supine, and it moves slightly to the left when he lies on that side. It is somewhat altered by distention of the stomach and by flatulence. It is more influenced by breathing. During deep inspiration the heart descends, the forward movement of the expanding left lung pushes it backwards and to the right, causing the apex to move towards the epigastrium. During forced expiration the heart descends and approaches the chest wall, a larger portion of its anterior surface is uncovered, the impact is more diffused and weightier; hence expiration

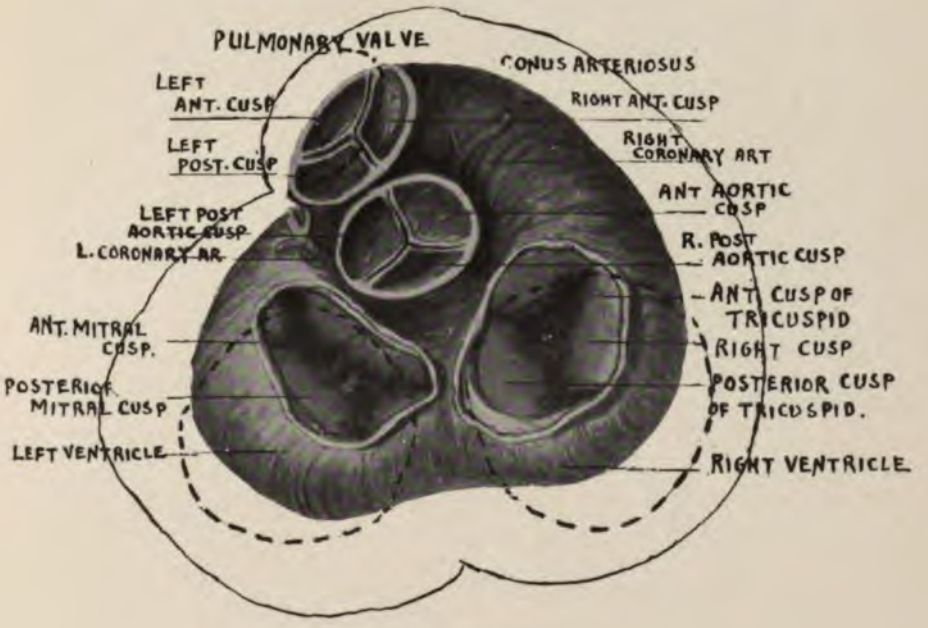


PLATE VI.

The Valves of the Heart, their Leaflets and Relative Positions. (After Murray.)
The outer line represents the heart in diastole, according to Spalteholz.

offers the most favorable time both for observation and for auscultation of this portion of the organ. Exercise and emotion render the impulse more distinct; wasting and debilitating diseases, degenerations and anæmias lessen it.

Not only do we mark the location of the apex, but we search carefully for pulsations in other regions, especially in the epigastrium and over the area of the right ventricle. The pulsations of the right ventricle are not visible in health, but are brought out by violent exercise, and diseased conditions similar to those mentioned as affecting the left ventricle may cause a permanent visible impact. The presence and character of this impact should be noted carefully, as in disease it is of considerable diagnostic value. Holding the breath until one begins to experience a sense of suffocation will bring about a condition exactly simulating hypertrophy of the right ventricle. The apex beat will disappear, and the impact of the right ventricle will be seen plainly in the epigastric area. Breathing being re-established order is quickly restored. Pulsations, arterial or venous, are often seen at the root of the neck and in the abdominal regions.

CHANGES IN THE POSITION OF THE HEART, THE RESULT OF DISEASE.

A pericardial effusion pushes the heart upward and obscures the apex beat. Even a slight degree of emphysema renders it invisible. Enlargements of the liver tilt the heart upward and outward. Hypertrophy of the right ventricle prevents the apex from striking against the chest wall, hence rendering it invisible. In cases where there have been previous attacks of pericarditis with the formation of adhesions the heart systole may cause at each stroke a more or less extensive retraction of the chest wall over the apex region, varying with the extent of the attachments. In children the whole præcordia may be yielding. In very extensive hypertrophy of the left ventricle the apex beat may be pushed downward and to the left, appearing in the sixth, seventh, or even the eighth, interspace, and several inches outside the nipple line, as occurs in the so-called *cor bovinum*. In slighter degrees of hypertrophy the area of impulse is displaced to the left in proportion to the extent of the change. Dilatation increases the area of pulsation and renders it more diffused without causing much displacement. In dilatation and fatty degeneration the impact is weakened; in hypertrophy it is strong and heaving.

INSPECTION.

Upward Displacements of the apex are often the result of diseases below the diaphragm causing pressure upon that structure. Such are solid and cystic growths of the abdominal organs, ascites and, to a lesser degree, tympanites. The upward dragging of the heart which results from phthisical contractions of the left lung is described under Pulmonary Tuberculosis.

Collections of air and fluids in the pleural cavity are important causes of displacement of the heart. Occurring on the right side, they are less potent than when found on the left. A large left-sided effusion may so displace the heart that the impulse is seen on the right of the sternum, even under the right nipple. It must be understood that the visible impulse in such cases does *not* correspond to the *apex*, but to some portion of the right ventricle which impinges against the thoracic wall. This condition has received the name of *dexiocardia*. Adhesions may cause the displacement to become permanent. Such a pulsation should not be confounded with that resulting from hypertrophy of the right heart, already mentioned, which occurs without displacement of the apex, although the apex beat *may be invisible*. In some cases of considerable hypertrophy of the right heart a widely diffused pulsation or heaving is seen over the entire epigastrium. It results from the exaggerated impulses communicated to the left lobe of the liver. It should be distinguished from similar pulsations communicated to the liver by the aorta, which can usually be done by timing the stroke with the fingers pushed under the edges of the ribs, where the heart contractions often may be felt. The aortic contraction is slightly later in point of time. In rare cases a pulsation is seen to the left of the sternum, at about the second intercostal space, which point corresponds to the root of the pulmonary vessel. Its area is limited, and on palpation a sudden, snapping recoil is felt to succeed the dilatation. The expansion is caused by the filling of the pulmonary vessel; the recoil by the closure of the semilunar valves. This condition occurs in cases of pulmonary phthisis in which there is advanced emaciation associated with a very considerable retraction of the left lung, and is comparatively infrequent.

Pulsation at the Root of the Neck, when seen in the supra-sternal notch, usually means aneurysm or dilatation, either of the aorta, which curves backwards less than an inch below the upper edge of the manu-

brium, or of the innominate vessel, which approaches the median line behind the top of that bone. Farther to the side lie the innominate veins; the left innominate vein traverses the root of the neck immediately below the sterno-clavicular junction, and often receives transmitted pulsations from the arteries. The jugular veins are frequently the seat of pulsations, either venous or transmitted. Venous pulsation is present in valvular disease of the right side of the heart. The condition, however, occurs independently of such disease and simply indicates venous regurgitation, with inefficiency of the *valves within the veins*, since a certain amount of regurgitation into the cavæ is physiologic. Venous pulsation is irregular and wave-like, and can be distinguished readily from the rise and fall of transmitted pulsations by making just sufficient pressure on the proximal side of the pulsation to obliterate the vein. Transmitted pulsations continue while the venous throb disappears.

Some of the phenomena just described are not wholly apparent to unaided inspection as they require additional diagnostic processes for their appreciation, but owing to their intimate relationship it seems simplest to group them together.

PALPATION.

Palpation for the most part confirms inspection, but the touch distinguishes some phenomena which are invisible. To elaborate all the conditions would be repetition.

The diversified alterations in the character and force of the heart's impulse are more readily appreciated by palpation than by inspection. An impulse can be felt which cannot be seen, and by touch its point of greatest intensity is more accurately fixed. We study changes in the rhythm and force of the beat, its character, its extent, and the presence of abnormal pulsations.

Temporary changes of both rhythm and force are produced by excitement, emotion, exercises or by hysteria. Permanent alterations are caused by hypertrophy, which increases the force of the impulse, and by dilatation, which enfeebles it while it increases its area. Morbid conditions which induce softening, such as acute infectious diseases, and general febrile states, enfeeble the impulse. Valvular diseases, adhesions, and displacements alter permanently the character as well as the position of the beat.

Effusion into the pericardial sac enfeebles, obscures or obliterates

the impact, and if perceptible it may have an irregular, wavy character.

Reduplication of the systole indicates pericardial adhesions.

Fremitus and a peculiar thrill, likened by Laennec to the purr of a cat, are sometimes noticeable. Both of these phenomena are interesting. The first, pericardial friction fremitus, may be felt at any point over the præcordial region in pericarditis. It is due to the rubbing together of the two roughened pericardial surfaces. It is superficial, and its character is harsh, rubbing or grating. The second, "the purring tremor of Laennec," is felt only at the apex or at the base, and is associated with valvular lesions. At the apex it is most characteristic when associated with mitral stenosis. The purr is then presystolic, but it may be present in roughening of the valves with regurgitation. (It is further discussed under lesions of the valves.)

At the base it is indicative of aortic valvular disease, or of aneurysm of the first part of the arch.

Congenital stenosis of the pulmonary valve is said to be associated with a similar tremor.

The sphygmograph and the cardiograph are mechanical aids to palpation which will be described later.

PERCUSSION.

By percussion we estimate the size of the heart as a whole, and the extent of the part of the organ which is uncovered by the lung. In an examination it is helpful and convenient to outline our results upon the chest with an aniline pencil. While such a diagram made by two examiners would be apt to vary, yet each might, none the less, draw correct inferences therefrom. If it is desired to ascertain the area of *relative* dullness, or that area which most nearly coincides with the true outline of the heart, then strong, firm percussion is employed; whereas if it is desired to ascertain the area of *absolute* dullness, which most nearly coincides with the uncovered area, then the slightest possible stroke is used. Since in expiration the heart approaches the chest wall, advantage should be taken of this for the determination of its size, but in outlining the uncovered area it should be borne in mind that the object of the examination is not to ascertain how much of the heart the patient may clear by pulmonary gymnastics, but to ascertain the existing conditions; hence the percussion should be made during quiet respiration. To obtain accurate results in

either case is by no means simple. The thin edge of the lung overlying the denser organ beneath so modifies the note that much confusion arises; but patience and practice bring rewards. Personally, we have found that the most satisfactory mode of determining the larger outline is to strike with just sufficient force to bring out clearly the normal pulmonary note, and to observe the slightest change of that sound as indicating the heart's border.

PERCUSSION AREAS OF THE HEART.

The thin, free edge of the right lung extends inwards to the middle of the sternum, and completely covers that portion of the heart which lies to the right of the mid-sternal line. Owing to the recedence of the left lung from the sternum, along the lower border of the fourth costal cartilage, which it closely follows, an irregular triangular area of the right ventricle is exposed as follows: base, the lower border of the fourth rib outward to the parasternal line (a vertical line drawn parallel to the mid-clavicular line, half way between it and the mid-sternal line). The perpendicular of the triangle is the left border of the sternum. The third side is a line drawn through the upper portion of the xyphoid cartilage and passing nearly, if not quite, horizontally outwards to the parasternal line, or to the apex. Practically it is very difficult to map out the third side of this area, since it is continuous with the upper border of liver dullness, and the differentiation between these two is very slight; but it may be said that the heart note lacks the absolute flatness elicited by percussion over the liver, where also the finger encounters a greater amount of resistance. Hence in percussing from below upwards, the fall in pitch and the dminution in resistance enables one to say that the heart has been reached. The method is sufficiently accurate for all practical purposes. When still greater exactness is required auscultatory percussion may be employed, with the aid of the Bowles instrument, whereby the differences above set down are brought out more clearly. The completed triangle is called the area of positive cardiac dullness. Its dimensions are, base 2 inches to 2½ inches (6 cm.); perpendicular 2 inches (5 cm.); hypothenuse 3½ inches to 4 inches (10 cm.).

Percussion affords the surest means at our command of estimating the actual size of the heart, but it will be gathered from what has been said that the method falls short of perfection. Hence, the area

as mapped out by this means, while indicating generally the heart boundaries, falls within its actual limits owing to the curvature of the organ and its consequent recedence from the nearly flat surface of the chest, just as the periphery of a sphere recedes from a plane on which it rests.

We begin on the left side, in the second interspace, as far outward as the parasternal line and percuss downward along this line until a change of the note tells us that we have reached the left upper margin of the heart, which should be in the third interspace. We follow this line of dullness inward to the sternum and pencil it as the base line of the organ. Crossing to the opposite side and percussing from right to left, in the same interspace, we obtain our relative dullness at the right border of the sternum, to which point we continue our base line. The line on the right may be traced downward along the right edge of the sternum as far as the fifth interspace. From the point of starting the left boundary line passes downward, with a slight outward curve, to the apex. The lower border line corresponds to the line of separation of the heart and liver, and sufficient has already been said regarding its determination. Forced expiration enlarges the boundaries by about one-half inch, the transverse diameter being more affected by respiration than the vertical.

The Area of Absolute Dullness. Hughes Bennett gives the dimensions of this area as indicated by the smaller figures above cited, saying that if these measurements are exceeded the heart is enlarged. He places the normal transverse diameter at 2 inches. This is certainly too small for well-developed men. I have found that even the larger area is often exceeded without any evidence of hypertrophy being evinced by the cardiac sounds or by changes in the arterial tension.

In women, owing to the interference of the mammary gland, and the change in the chest contour from corset-wearing, these limits are harder to define and are subject to much broader variation than in men. In children, careful percussion often fails to outline satisfactorily either the area of relative dullness or the area of absolute dullness. Even when the latter can be defined its dimensions are proportionately smaller than in the adult.

CHANGES IN THE AREA OF PRECORDIAL DULLNESS.

A full inspiration diminishes the area of dullness by displacing the heart backwards and extending the lung forward. Changes

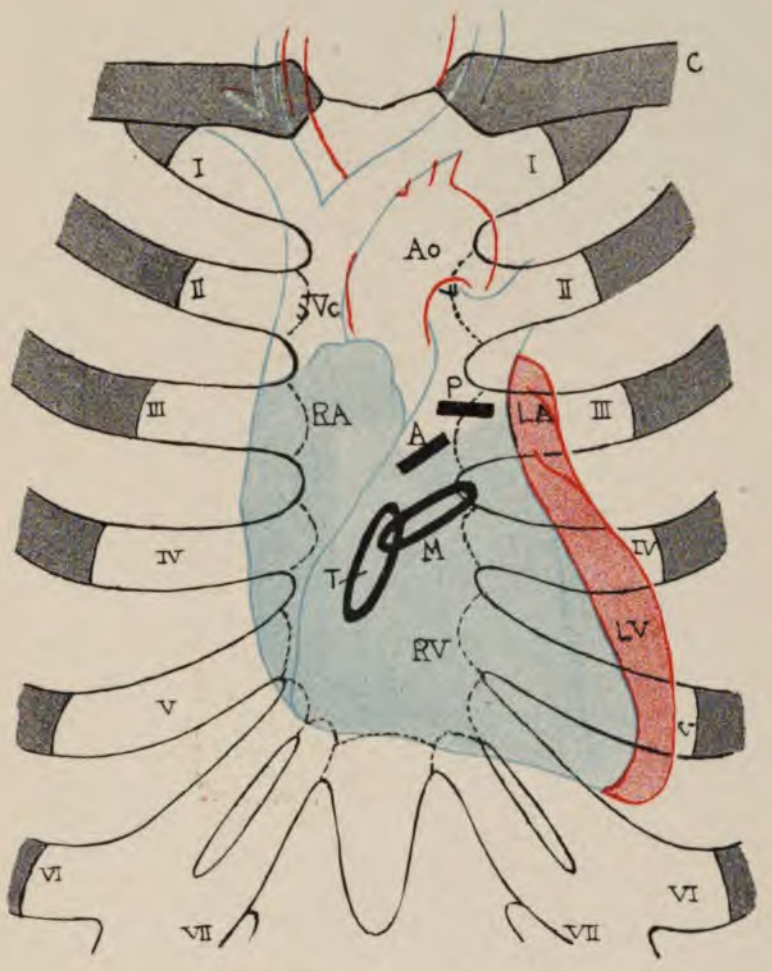


PLATE VII.

Relationship of the Cardiac Area to the Costal Cartilages and Sternum.

RV. Right Ventricle. LV. Left Ventricle. RA. Right Auricle. LA. Left Auricle. S.Vc. Superior Vena Cava. Ao. Aorta. P. Pulmonary valve. A. Aortic valve. M. Mitral valve. T. Tricuspid valve.

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wrought by disease are: an increase, a diminution, or a displacement of the dull area.

Decrease in its area may result from cardiac atrophy which accompanies all protracted febrile and wasting diseases, but such atrophy is generally inappreciable. The more usual cause of decreased area is disease of the lung—emphysema being the chief offender. Even a slight degree of emphysema affects the marginal air-cells.

Hypertrophy of the heart is the most frequent cause of *increase* in the præcordial area. Hypertrophy of the left ventricle enlarges the area outward, to the left, and somewhat downward, so that the apex beat may be found in the sixth or seventh interspace. Hypertrophy and dilatation of the right side increase the percussion area to the right of the sternum. In cases of hypertrophy or dilatation of *both* ventricles the transverse distance is increased on both sides of the bone.

The other phenomena accompanying and confirming the above condition are described under their separate heads.

Effusions into the pericardial sac produce a lateral extension of the percussion dullness, but therewith is a cone-shaped *upward prolongation* of the dull area towards the left sterno-clavicular junction. It is reiterated that hypertrophies do not materially increase the vertical area of dullness. Effusions impart an increased sense of resistance on percussion, and sometimes there is a filling or lifting of the præcordia with each impact.

Tumors of the mediastinum and aneurysms increase the dull area. The diagnosis of aneurysm is given with the description of the disease. From new growths the distinction lies in the fact that there the heart sounds are unchanged in character and are heard only over their normal areas, and are *not co-extensive with the increased area*, which would be the case were the heart itself enlarged; and that the inspection signs of enlarged heart are absent. Cancer gives cachexia. The history, the dyspnœa and the subjective signs all aid in the separation of the conditions. Consolidation of lung tissue overlapping or adjacent to the heart may mislead. Auscultation will generally settle the difficulty.

Displacements of the Area of Dullness occasionally result from tumors. Emphysema causes a slight displacement as well as decrease of area. Effusions are the most potent causes of displacement, especially when they are into the left sac. (For diagnosis see Pleu-

ritis.) Air in the pleural sac operates in a like manner. (See Pneumothorax.) Displacements due to infra-diaphragmatic causes are listed under palpation in the preceding section.

AUSCULTATION OF THE HEART.

When the ear or a stethoscope is applied over any part of the chest of a healthy individual the heart sounds are heard with varying distinctness. If these sounds are analyzed they are found to consist of two distinct and easily recognized components, separated from each other by an interval of silence. While each of these sounds varies in *intensity*, as we change the position of the stethoscope, yet each is audible over the entire cardiac area, and preserves its distinctive *quality* throughout. It is difficult to graphically represent these sounds, and no better way has yet been suggested than the time-honored one of imitating them by pronouncing the two monosyllables, "lub" and "dup." They represent respectively the first and the second sounds of the heart.

The first sound, lub, is best heard at the apex, where its intensity is greatest, and where it is not only louder than the second, or dup, sound, but is of longer duration than that sound as well as more booming or resonant in character and lower in pitch. The second sound is best heard immediately over the sternum, opposite the second interspace. It follows from what has been said that this sound is higher in pitch, shorter in duration, having less of a boom and more of a sharp, cut-off character at its termination than the first. And although the first sound listened to *at this point* is less prolonged and less weighty than it is at the apex, yet at whatever part of the chest they may be heard the healthy second sound is shorter in duration and higher in pitch than is the first.

The two sounds present further modifications when listened to over the right ventricle and over the pulmonary artery, but in the main they preesrve the characters already attributed to them. The character and modifications of the sounds are best learned by experience, since a wordy attempt to describe minute differences is confusing rather than helpful. When we examine into the mechanism of the sounds we can still better appreciate their character.

The principal element in the production of the first sound is the closure of the auriculo-ventricular valves, but along with this sound and modifying its quality must be named the apex impact, the note of

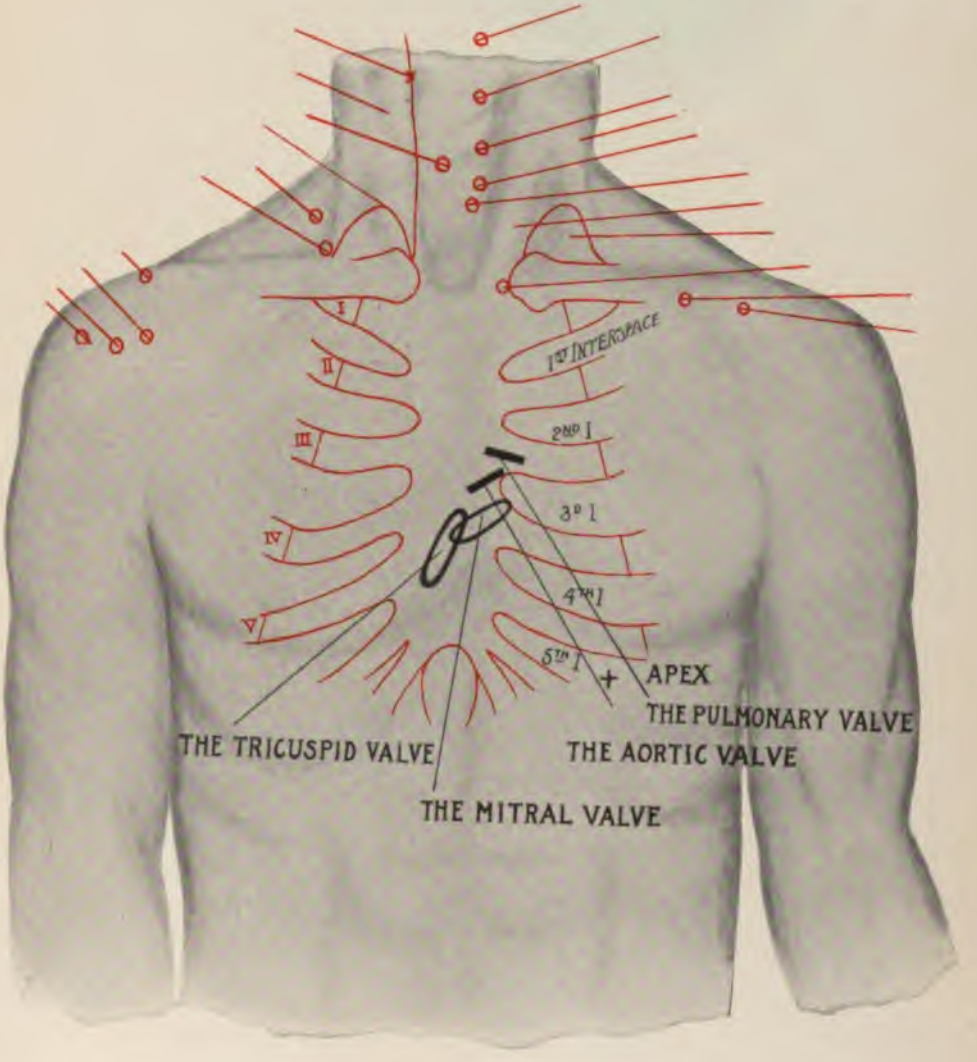
1ST INTERSPACE
2ND I
3D I
4TH I
5TH I
APEX
THE PULMONARY VALVE
THE AORTIC VALVE
THE TRICUSPID VALVE
THE MITRAL VALVE

PLATE VIII.

muscle contraction, and the noise made by the rush of blood through the aorta and the pulmonary arteries. Were it not for these modifyers, neither its quality nor its duration would differ from that of the second sound, which is produced solely by the closure of the aortic and pulmonary semilunar valves, caused by the reflux of the blood column upon their leaflets; hence almost the sole variation to which the second sound is subject in health is diminution of its intensity as we recede from the point where it is produced. As has been stated, the two sounds are separated by a pause, and a second interval follows the completion of the heart beat, this second pause being approximately three times as long as the first.

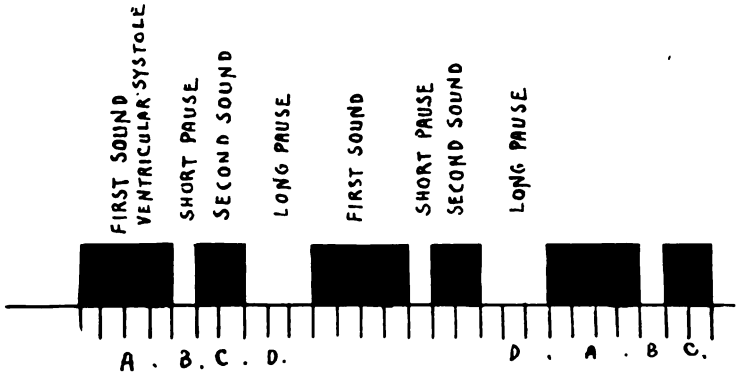

Fig. 39—Auscultation of normal heart sounds. Relative length of normal heart sounds drawn to a scale of 1-10. Each division below the line represents 1-10 second.

While the normal heart rate for adults is upwards of seventy per minute, yet for convenience we may assume a rate of sixty beats per minute, since this rate greatly facilitates the description. Then one beat or complete heart cycle will occupy one second. If now we divide the complete cycle occupying one second into tenths of a second, the relative length of the factors which make up the cycle will be, first sound, 4-10ths, short pause, 1-10, second sound, 2-10ths, long pause, 3-10ths; total, 10-10ths. This is indicated by the accompanying simple diagram.

A—Ventricular systole.

B—Short pause.

C—Closure of aortic and pulmonary valves.

D—Ventricular diastolé.

As said, the long pause or period of rest varies with the heart's rate. When the heart's pulsations follow each other with great rapidity, the period of rest is reduced to the minimum, and when the heart's action is slow the period of rest is lengthened and becomes longer than the period of action. On the other hand, when beating rapidly the long pause so lessens as to be shorter than the period of action, hence the normal sounds of the heart may seemingly change their relationship to one another, and, when the action becomes very rapid, to the listener the *first* sound seems to *follow* the *second* sound, since the interval betwen the *second* sound and the recurrent *first*

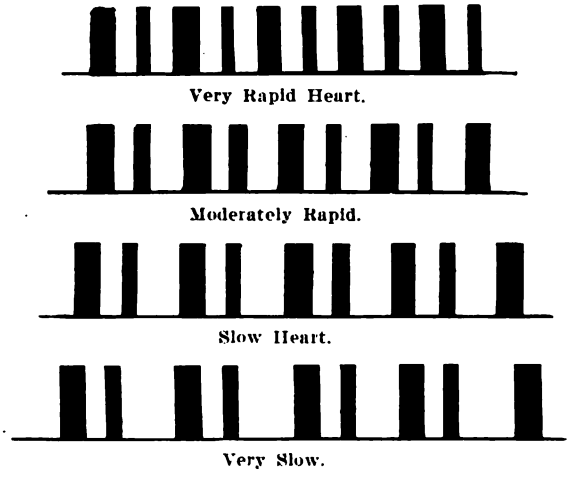

Fig. 40—Diagram illustrating the relationship in time of the first and second heart sounds in different rhythms.

sound is now longer than the interval between the first sound and the second; a state of affairs which may be embarrassing to the student.

In the slowly-acting heart the distinction is easy, since it is only necessary to remember that the long silence follows the second sound, but in cases of rapid, irregular action accompanied by a change in the qualities of the two sounds which makes them resemble each other, or when only the first sound is audible at the base and the second only audible at the apex, the difficulty of separation is considerable.

The accompanying diagram may aid in comprehending the relationship.

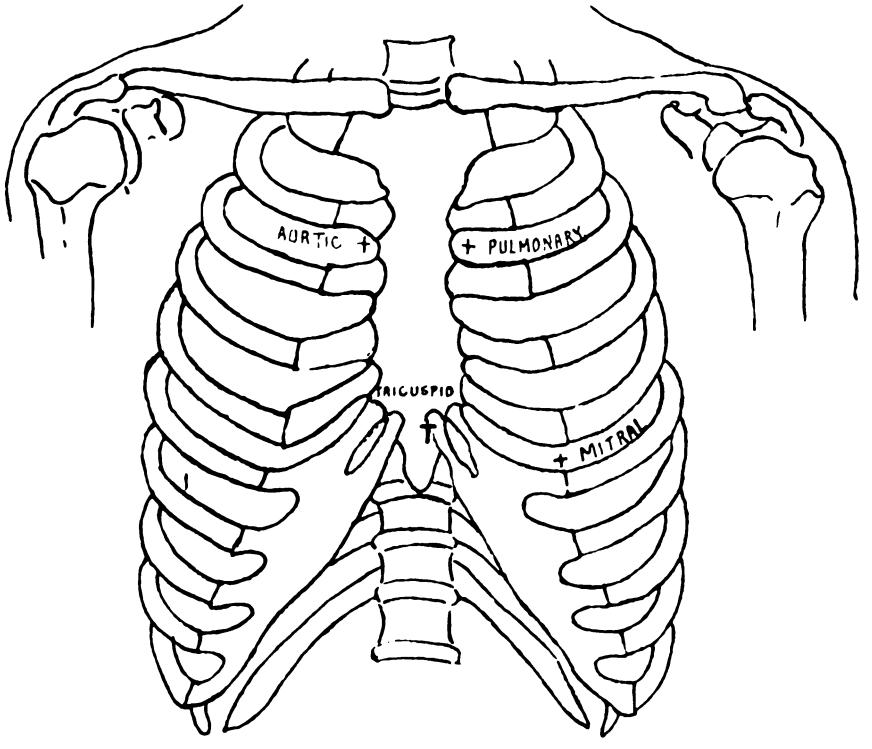

PLATE IX.

The cardinal points of the valve areas. Points at which valve sounds are best isolated.

POSITION OF THE VALVES OF THE HEART.

The valves are all located within such a narrow radius that a silver quarter may be so placed as to cover a portion of each. Indeed, the aortic and the pulmonary semi-lunar valves are separated from each other only by the thickness of the aortic wall. The two auriculo-ventricular valves and the aortic valve are in one plane, which forms a diagonal of the thorax. As described in the various text-books, much variation in the location of these valves will be found to exist. Most recent anatomical research as to their exact topographical position locates them as follows: The pulmonary valve lies beneath the third left costal cartilage, but close to its junction with the sternum, and is the most superficial as well as the highest up of all the valves.

Were the position of the heart vertical the aortic valve would lie almost exactly behind the pulmonary valve, but, viewed from in front, the heart is the subject of a double tilt. The vertical axis points from right to left and the antero-posterior axis points from above downward. For this reason the aortic valve occupies a lower level than the pulmonary valve, at the same time being deeper-seated, lying behind the left edge of the sternum, opposite the upper part of the third interspace.

The mitral valve, the most deeply placed of the four, lies beneath the left half of the sternum, opposite its junction with the fourth cartilage. The right auriculo-ventricular, or tricuspid valve, is more superficial than either the mitral or the aortic valves; its aperture is almost vertical, and it lies under the middle of the sternum, opposite the fourth interspace. The upper, inner segment of its opening overlaps the lower inner segment of the mitral orifice. It will be seen, therefore, that all of the valves lie under the middle and left half of the sternum, between the upper border of the third cartilage and a short distance below the fourth.

ISOLATION OF THE VALVE SOUNDS.

It might strike the student as a difficult, if not an insurmountable, task to isolate the sounds arising from four structures so nearly superimposed. Such, indeed, would be the case were it not that the points of isolation, the point at which each separate sound is best heard, does not correspond to the surface area above given, nor even to the superficial point nearest to the actual seat of the valve. This is due to the fact that the sounds emanating from the valves, made by

the impinging of the blood current against their leaflets and from their closure, is best propagated in the direction of the blood current; hence the apparent discrepancy. The locality in which the mitral sound is best heard is over the apex area. The tricuspid is best heard close to the left border of the sternum, at the fifth interspace, or over the xiphoid cartilage. The aortic valve sound is loudest and clearest, and should therefore be listened to, on the right of the sternum in the second interspace, where the aorta approaches closest to the chest wall, for which reason the second right costal cartilage is named the aortic cartilage. On the opposite side of the sternum (left), in the same interspace, is best heard the pulmonary valve. This gives us four cardinal points at which the four sounds are best isolated, at which their individual qualities are strongest and easiest to differentiate.

INFLUENCES WHICH CAUSE ALTERATIONS IN NORMAL SOUNDS.

Since inflation of the lungs increases the thickness of the heart cover, diminishes its exposed area, as well as causes recession of the organ, all the sounds are better heard during forced expiration. Advantage should therefore be taken of expiration to differentiate sounds which are otherwise inaudible or only feebly heard. The normal sounds of the heart are best heard with the patient sitting or standing, although its areas are best mapped out with the patient lying on his back. But in case of suspected disease of the organ it is absolutely essential to percuss and to auscultate the patient both while *standing* and while *lying down*. The diagnosis may be materially influenced by this precaution, and doubtful cases may be removed to the realm of certainty by the procedure. Thus, a murmur may entirely disappear when the patient lies down, and reappear when he rises. It may confidently be stated that such a murmur is not organic. On the other hand, a murmur which is only audible when the patient is in the recumbent posture is probably organic.

CONDITIONS WHICH AFFECT THE INTENSITY OF THE SOUNDS.

The normal heart sounds are heard with greater intensity, as well as over a larger area, in persons whose chest walls are thin; also as the result of excitement; in cardiac hypertrophy; during the sthenic stage

of fevers; as the result of certain cardiac stimulants; and as the result of increased density of adjacent pulmonary tissue. As a result of the last-named, they may be conducted to very distant points. The full significance of this sign is considered under the section on Phthisis.

Conversely, the heart sounds are rendered weaker even in health by abnormal causes. Fear or fright so acts, as do debility, wasting diseases, and the anæmias. Many drugs have the power to depress the heart, and careful inquiry should be made as to whether the patient is addicted to drug habits. Diseases such as fatty degeneration of the muscles; mechanical causes interfering with cardiac action,—as effusion into the sac of either the lung or the heart,—produce like results. Not infrequently one encounters a heart whose sounds are abnormally weak and inaudible without any assignable cause, the subjects being generally women.

Except in cases of cardiac hypertrophy, or when consolidation of tissue has taken place, the extent of the area over which the sounds are audible has no special significance. Full inspiration obscures certain of the sounds. This is particularly true of the pulmonary valve sound heard on the left side in the second interspace. Likewise during full inspiration the aortic sound is lightened in intensity. The apex sound is now best heard nearer the median line, as the apex is moved in that direction by the lung expansion. Nervous excitement and exercise temporarily increase the intensity of both the heart's action and its sounds. Such action is named palpitation. Advantage may be taken of exercise to temporarily intensify the sounds when auscultating a heart whose action is feeble. Softening of the walls diminishes the intensity of both the sounds, particularly the first. This is observed in many acute febrile and infectious diseases, and in the degenerations. Thickening of the walls changes the quality of the tone, especially of the first sound, lengthening it and rendering it more heavy and booming, thus lowering the pitch of its note. This is the result of hypertrophy, however brought about.

The quality of the second sound is more constant, and changes materially only in disease of the valves or in grave alterations in the composition of the blood. Increased back pressure on the valves causes a forcible, snap-like closure, which augments the second sound both in clearness and intensity. This is especially the case with the pulmonary second sound, and in lung consolidation is an important

sign of beginning of pulmonary infiltration. Aneurysm of the aorta will cause accentuation of the second sound of that valve.

In case of fluid in the pericardial sac both sounds seem to come from a distance and to have a feeble, muffled character. Other changes worthy of notice are the reduplication of one or the other sound and the lengthening or shortening of either interval, the long or the short, between the sounds.

Reduplication. Generally it is the second sound which is repeated. This curious phenomenon can be explained only on the hypothesis that the corresponding valves, the aortic and the pulmonary on the one hand, and the mitral and the tricuspid on the other, fail to close synchronously; or else on the assumption that the two allied chambers of the heart fail to contract in unison. Such a theory as the first is very plausible and could be brought about by any relatively important change in the blood pressure of the two circulations, the pulmonary and the systemic. From the nature of the case, the former more often will be at fault, and pulmonary engorgement by increasing the back pressure on the doors of the pulmonary artery will cause that valve to anticipate slightly its time of closure, and, indeed, such reduplication often occurs after running, as is seen in sprinters. In the same way narrowing of the blood channels, the effect of sclerosis of large organs, particularly the liver or the kidneys, would produce similar results on the aortic valve.

Retardation in the closure of the tricuspid valve causes reduplication of the first sound.

Reduplications are sometimes intermittent, sometimes rhythmical, that is, they skip a certain number of beats, recur at stated intervals, skip and recur almost melodiously.

Shortening of the silent intervals in relation to pulse rapidity has already been referred to.

ADVENTITIOUS SOUNDS.

The normal sounds of the heart may be replaced by or be intermingled with other sounds adventitious in their nature, totally different in quality from the normal sounds. The recognition of these sounds and the correct interpretation of their relation to the heart's mechanism, are of prime importance in the diagnosis of the many ailments to which the structure is liable. These new sounds are classified as *pericardial* or *endocardial.*

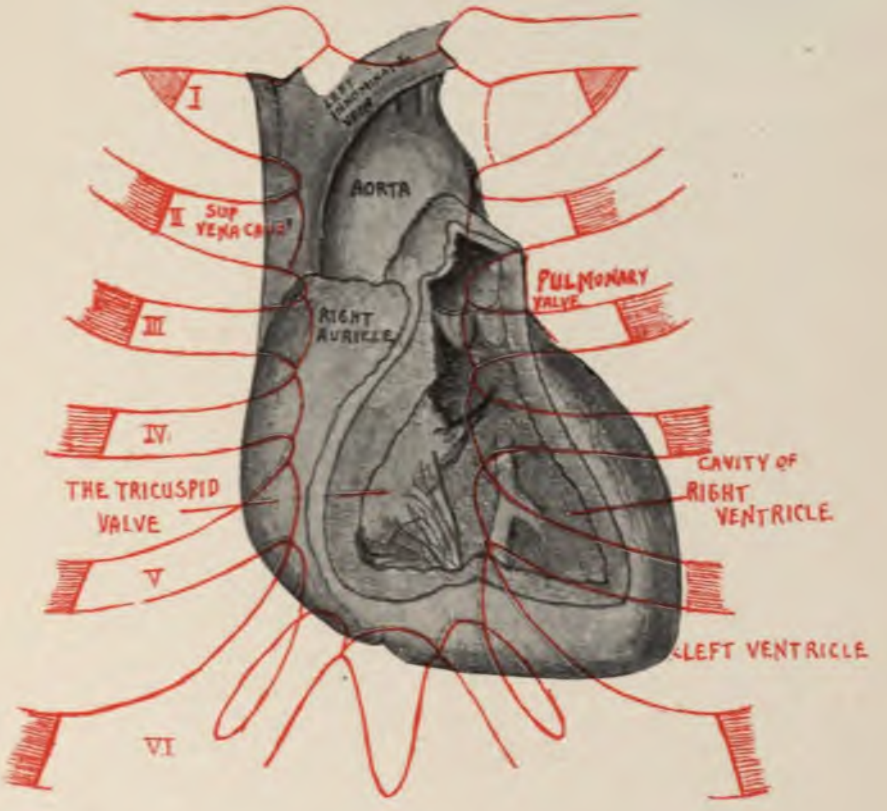

PLATE X.

THE TRICUSPID AND PULMONARY VALVES.

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Those sounds which are due to alterations in the structure or changes in the condition of the adjacent layers of the pericardial sac, namely, the visceral and the parietal layers, are termed *exocardial* or *pericardial murmurs*. Such changes are usually the result of inflammation, but may be caused by external pressure. From their character they are called *friction sounds*. The manner of their production will be considered hereafter.

Those sounds that originate from changes occurring within the organ, which are essentially changes affecting those reduplications of its serous lining which form the valves, are classified as *endocardial*. In contradistinction to the friction sounds they are termed *murmurs*.

Endocardial murmurs vary extremely in character, pitch, tone, duration, location, time, and in the distance and direction in which they are transmitted. They often receive their names from a fancied resemblance to some sound in nature: hence, we read of blowing, cooing, rustling, clacking, rasping, grating or filing murmurs. Such terms are useful since they generally roughly indicate the pitch. The majority of murmurs are soft and blowing, yet occasionally they are so harsh and noisy as to be audible to the bystanders.

CAUSES WHICH PRODUCE MURMURS.

As to the exact physical or mechanical causes which operate to produce the above-described sounds we have no positive knowledge, but a plausible theory may be formed by reasoning from existing pathologic conditions. We know that murmurs accompany grave changes in the composition of the blood without any recognizable structural alterations of the heart, the valves, or the vessels. Such murmurs are called *hæmic* murmurs, and are described as functional or accidental. They are associated with most of the grave anæmias, but occur in other diseases as well. By some writers the term *inorganic* murmurs is applied to sounds so arising; both this term and the name nonorganic are objectionable in this sense.

On the other hand, murmurs are associated with certain pathologic alterations of structure which can stand only in a causative relation thereto. Enumerated they are:

(a.) Narrowing of the orifices through which the blood finds exit.

(b.) Widening of the orifices.

(c.) Narrowing or widening of the bloodvessels or heart chambers.

(Changes under a. and b. are in effect changes in the valves, such

as roughening, thickening, or puckering of the leaflets.—deformities resulting from sclerosis. Inflammatory deposits or vegetations result in perforations or contractions of the leaflets.)

(d.) Imperfect opening or closure of the valves due to causes mentioned and to shortening of the *chordæ tendineæ*.

It were profitless to discuss the laws of hydraulics and hydrostatics which might seem to apply. Suffice it to say that the noises are produced by the *vibrations* set up in both liquids and solids by the circulation of the blood over the altered structures. It is also conceivable that blood alterations may influence these murmurs, especially by increasing or diminishing their intensity.

Organic murmurs may be produced at any of the four valves of the heart, but by far the most common valve to be affected is the mitral; next in order of frequency is the aortic, then the tricuspid, with the pulmonary a bad fourth. The aortic and mitral valves together are affected four-fifths as often as the aortic valve alone.

RESULTS OF VALVULAR IMPAIRMENT.

Inability of a valve to close so as to render itself "water-tight" is called *insufficiency*, and the return flow of blood through the orifice is called *regurgitation*. The causes have been enumerated. It is, however, well established that the phenomenon of regurgitation may occur during excited and violent-acting states of the heart, when both valve and muscle are perfectly healthy. Such inefficiency can be explained only as a temporary expansion of the valvular ring or by imperfect closure, just as other automatic acts are disturbed by overstimulation. So, also, altered states of the blood may be accompanied by all the audible signs of regurgitation without organic change being present. No doubt in many of these cases the insufficiency is actually present, only to pass away with the improvement in the general condition.

Contraction, puckering, or thickening of the valve leaflets, or deposits upon their surface, shortening and thickening of the *chordæ tendineæ*, and deposits into the valvular fibrous ring, operate to produce such a narrowing of the valve opening as to interfere with the outflow of the blood. Such interference is called *valvular stenosis*. The aortic and the left auriculo-ventricular orifices are oftenest affected.

TIME OF CARDIAC MURMURS.

Having considered the physiologic action of the heart and the mechanism of the murmurs we are prepared to discuss the relationship of the latter to the former in point of time; in other words, to fix the rhythm of the murmur as regards those acts which constitute the complete heart cycle, viz., the systole, the diastole and the period of rest.

A murmur which accompanies only a single act of the heart, the filling or emptying of a chamber, the opening or closure of a valve,

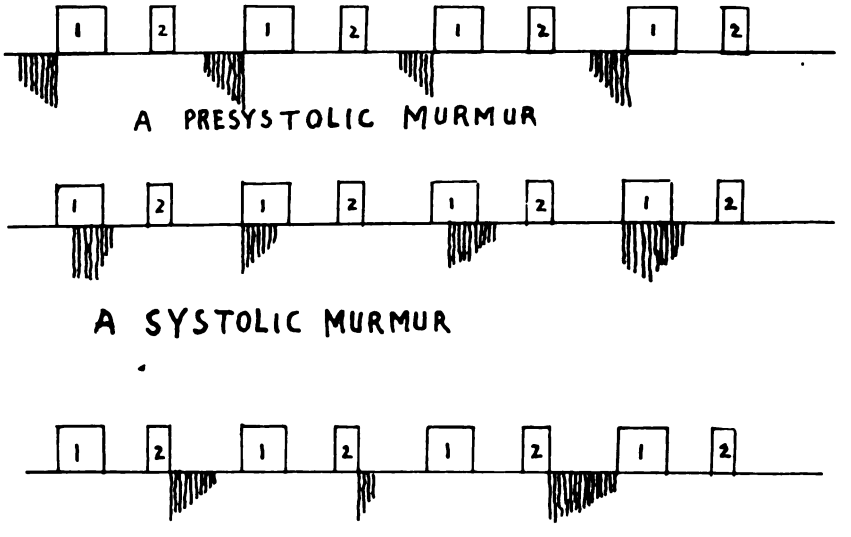


Fig. 41—Diagram illustrating the single or simple murmurs.

is classified as a *simple* or *single* murmur. Murmurs which arise in connection with two of these acts, constituting a union of the simple murmurs, are called *compound* murmurs. Compound murmurs arising at one orifice or valve are called *double* murmurs, when originating at different orifices they are called *combined* or *associated* murmurs.

Every simple valvular murmur has one of three relations to the sounds of the heart, one of three places in the cardiac cycle.

First, the murmur may be synchronous with the *contraction* of the *auricles*, in which case it is *auriculo-systolic*. In time such a murmur precedes the first sound, running up to that sound but ending

at the moment of its production. The relation which any murmur bears to systole or diastole may best be ascertained by placing the stethoscope over the seat of the murmur, and, while the tip of the finger records each systole, the attentive ear consigns the murmur to its proper period in the heart cycle.

Second, the murmur may be synchronous with the *contraction* of the *ventricles*, in which case it is *ventricular-systolic*. In point of time such a murmur follows the first sound of the heart and ends somewhere between the first and the second sound. Sometimes the murmur is so prolonged as to almost touch the second sound. It is quite apparent that in this case the murmur may actually begin with the beginning of systole, but will not be apparent until the cessation of the true systolic sound, hence will follow immediately upon that sound as though it were a prolongation of systole.

Third, the murmur may be synchronous with the *dilatation of the ventricles*, in which case it is *ventricular-diastolic*. In point of time such a murmur *follows the second sound* and ends during the interval betwen the second sound and the recurrence of the first sound.

Briefly recapitulated the simple murmurs are, therefore, presystolic, systolic, or diastolic in relation to the functions of the heart.

In accordance with the simple scheme adopted for representation of the heart sounds we may, by a slight modification, represent also the simple endocardial murmurs and their relations to the normal sounds:

Various combinations of the simple murmurs, constituting the compound or double murmurs, are not of rare occurrence, and greatly increase the perplexity of correctly timing them. They are illustrated in Diagram.

The most common of these associated or combined conditions, as we shall see, is the co-existence of a ventricular-systolic and a ventricular-diastolic murmur; then, of an auricular-systolic with a ventricular systolic murmur. In certain cases of long standing, in which two or more valves are affected, four or even six murmurs might possibly be present in the same heart.

LOCATION OF CARDIAC MURMURS. VALVULAR AREAS.

The point of origin of a murmur, the direction and the limit of its diffusion, are pretty constant quantities, and clinical experience combined with post-mortem confirmation have established certain fields

which are called the *areas of the murmurs*. The location or seat of the murmur is determined by finding its point of greatest intensity; by mapping out the field over which the sound is audible, the direction and extent of its transmission, in conjunction with the associated symptoms. Even when all these elements are weighed there is a possibility of error, and experience rather than precept must teach the student that the presence of an adventitious sound does not necessarily mean organic disease, nor does its absence indicate freedom from such a malady.

We examine successively the four cardinal points already named, at which normally the four valve-sounds attain their maximum in-

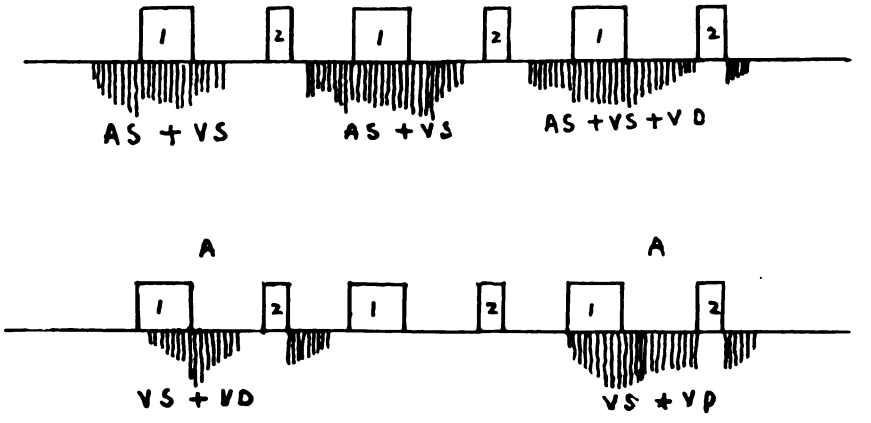


Fig. 42—The combined murmurs. AS Auricular systole. VS Ventricular systole. VD Ventricular diastole.

tensity (see plate). If a murmur is clearest and most distinctly heard at the apex, it is strong presumptive evidence that it concerns the mitral valve and the left ventricle. The mitral area is a small circle surrounding the apex of the left ventricle, as illustrated in the accompanying plate. Its location, therefore, changes with changes in the position of the apex, and enlarges with enlargement of the ventricle; hence the necessity of accurately locating the apical point. Mitral murmurs are more generally propagated to the left than to the right of the apex. Especially is this true of regurgitant murmurs. The obstructive mitral murmur is apt to keep within bounds, and its point of greatest intensity corresponds pretty accurately with the center of our circle—that is, it is a little to the right of the apex point.

A corresponding circle on the back, of which the inferior angle of the left scapula is the center, will often enclose a field in which are distinctly heard the mitral murmurs, especially in emaciated persons, and this field should not be overlooked. This is admirably shown in the photograph of the patient with mitral insufficiency, on page 209, in which case the murmur was transmitted directly to the area shown, without following around the axillary space.

Both DaCosta and Naunyn draw attention to the fact that some mitral murmurs have their seat of greatest intensity, not in the above area, but in the second or third interspace, an inch-and-a-half or two inches outside the left border of the sternum,—that is, just beyond the pulmonary area, for lesions of which valve they might possibly be mistaken. The murmurs thus centering are usually regurgitant, and the explanation lies in the fact that at the point indicated the auricle is most superficial, and that it crops out still further when dilated by the regurgitated fluid. Some of the murmurs assigned incorrectly to the pulmonary valve, are, according to Balfour, in reality mitral murmurs so caused. While the center of intensity is higher up in these cases than the usual mitral center, yet this should not deceive the cautious observer. Their area is outside the pulmonary area, and they are heard, but with less distinctness, in the mitral circle.

Tricuspid Area. If the murmur is best heard over or at the left margin of the ensiform cartilage where the right ventricle is exposed, it is highly probable that it emanates therefrom, and that the tricuspid valve is the malefactor. In case of considerable dilatation or hypertrophy of the right ventricle the valve moves towards the *left*, and the sound is heard along the sixth interspace, (sixth and seventh ribs) to the left of the sternum.

The tricuspid area is triangular in shape, larger in its bounds than the preceding area, since it lies more to the front. Its limitations are a line crossing the sternum from the lower border of the fifth right costal cartilage to the upper border of the third left cartilage, thence downward to the center of the mitral area above described, thence across the ensiform cartilage to the point of beginning. The diagram gives a clearer idea of the area than can be given by description. The left boundary coincides with the left interventricular cardiac groove.

Pulmonary Area. If a lesion of the pulmonary valve exists the auditory area is limited to a small circle over the origin of that artery

in the second and upper third left interspaces. The inner border of the circle touches the left edge of the sternum. These murmurs are superficial, hence distinct. The sound disappears when searched for across the right sternal border, but may be indistinctly transmitted upward toward the left sterno-clavicular articulation. It is *not* conducted into the great vessels of the neck, nor is it audible in the apex area. These limitations serve to separate it from both mitral and aortic murmurs. Organic pulmonary murmurs never arise in intercurrent diseases, although functional murmurs are often credited to this rôle.

Aortic Area. If the region in which centers the intensity of the adventitious sound be the upper sternal, especially if the region lie to the right of the bone, the murmur in all probability arises from a defective aortic valve. These murmurs are heard with little variation of their intensity beneath the manubrium, and on both sides of the sternum, in the third left and in the second right interspaces, which latter is just below the aortic cartilage, at which point aortic murmurs attain their maximum intensity, as a rule; but owing to the position of the aorta and the direction of its current they are audible, ofttimes, the entire length of the breast-bone, and in cases of regurgitation the center of intensity is not infrequently over the ensiform cartilage, and even extends over to the mitral area.

Aortic obstructive murmurs are transmitted into the vessels of the neck with considerable clearness and intensity, which fact distinguishes them easily from all other murmurs. Even regurgitant aortic murmurs may be heard faintly in the cervical vessels. The obstructive murmur is sometimes heard in the course of the great vessels down the spine and even in the extremities. The diagram shows the area of these murmurs as it appears on the anterior aspect of the chest.

MITRAL MURMURS.

When during the long cardiac pause the blood returns from the lungs to the left auricle, the inflow causes that chamber to dilate (auricular diastole). As soon as this auricle is filled, the chamber contracts, (auricular systole) and forces the blood through the left auriculo-ventricular orifice into the left ventricle. If now the mitral valve has been affected by disease in one of the ways set down, so that the orifice is narrowed and the flow obstructed, the current no longer passes, silently and swiftly, through the gateway to its ventricle.

The obstruction causes the emptying process to be impeded and therefore *prolonged*, and the passage of the blood is accompanied by a murmur, which has its greatest intensity in the apex region and which, in point of time, occurs during the auricular systole, that is, *before the apex impact*. Such a murmur denotes *mitral stenosis*, and is so named. It begins when auricular systole begins and ends with or before the completion of that act. It occurs while the blood is being expelled from the auricle and while the ventricle is still passive, but it frequently results in a prolongation of the auricular contraction. Being produced by the blood flowing in its natural direction it is spoken of as a *direct* murmur, in contradistinction to murmurs arising when the blood flow is in a direction contrary to its natural course, such murmurs being named *indirect* murmurs. Special attention is directed to the fact that this murmur may be distinctly audible only when the patient is lying down, disappearing when he sits or stands. Although this murmur occurs during ventricular diastole, it is not to be understood that it is audible during the entire period of the ventricular dilatation, although blood is flowing passively into the cavity during the whole of that act. Ventricular diastole closes with auricular systole, and the added impetus given to the current by this contraction of the auricle may be necessary to impart sufficient force to the stream to produce the murmur. The auricular contraction is inaudible, but is immediately followed by the ventricular contraction, which produces the first sound of the heart. Since the murmur is generated immediately before this latter contraction or systole, it is called *presystolic*, which distinguishes it from those murmurs whose duration is that of the entire diastolic period. The special point regarding its *tempo* is that it is not heard immediately after the *second* sound of the heart, but immediately before the *first* sound.

With or without encountering obstacles in its path the blood from the left auricle now reaches and distends the left ventricle (ventricular diastole), whose contraction immediately succeeds its distention (ventricular systole). In the healthy heart the only escape for the current imprisoned by the closure of the mitral gate is through the open aortic valve. If, however, disease has wrought changes which inhibit the perfect closure of the mitral leaflets, when the blood surges against them during the ventricular contraction they will not suffice to stop its flow, but will permit a portion of the fluid to pass backwards and re-enter the left auricle. This back-flow is further aided by

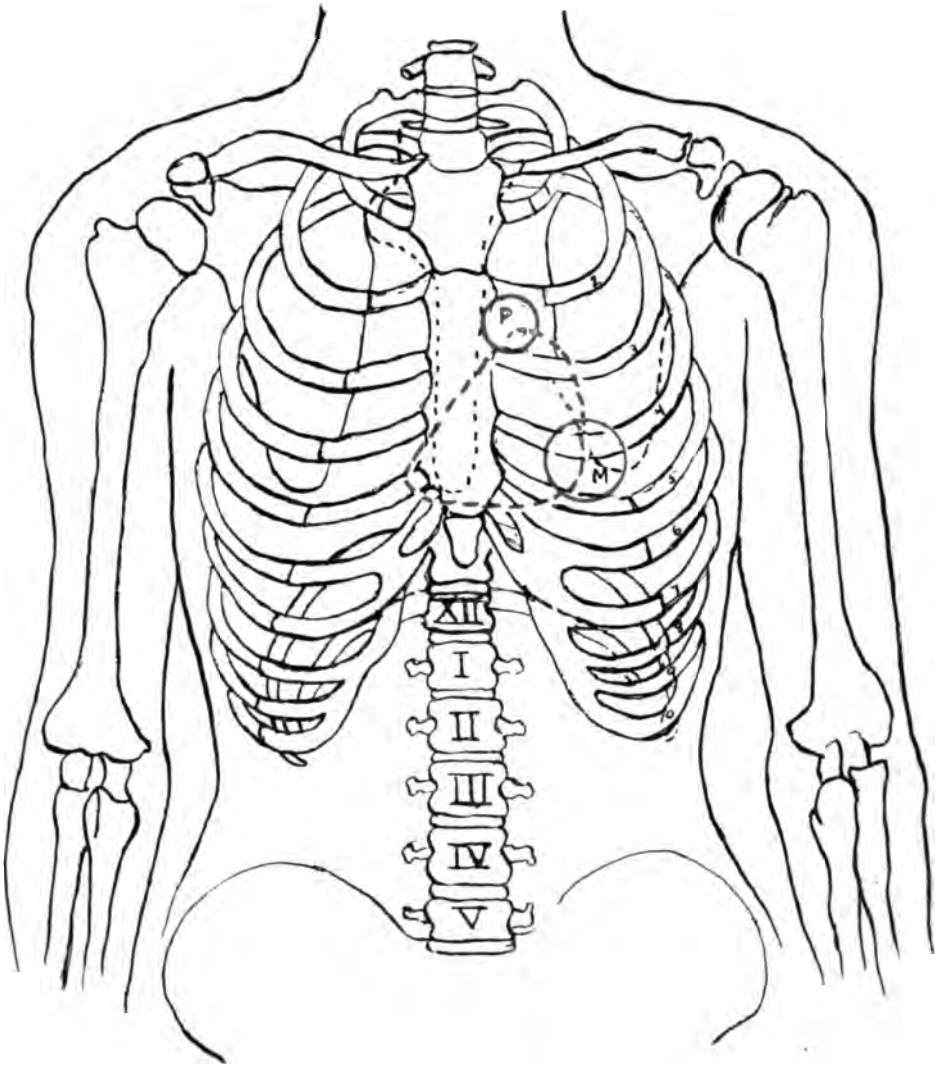


PLATE XI.

THE VALVULAR AREAS.

Red Circle, Mitral Area.
Triangle, Tricuspid Area. P. Pulmonary Area.
The Aortic Area in black dots.

the slight negative pressure existing in the auricle during that period of the cardiac cycle. The valve permitting this return flow just described is said to be *incompetent*, or *insufficient*, and the reverse current is called *regurgitation*. In this case it is mitral regurgitation. This phenomenon is indicated also by an accompanying murmur, which, in its beginning, is synchronous with ventricular systole,—that is to say, the apex impact and the carotid pulse, and accords with the first sound of the heart; or it immediately succeeds that sound and prolongs it, ending between the first and second heart sounds. It is the most common of the valvular defects. The preceding murmur may exist as an independent entity, but is more often associated with the present one.

TRICUSPID MURMURS.

The right ventricle acts in unison with the left, its systole and diastole are at one with its fellow. The tricuspid valve bears the same physiologic relation to the right heart that the mitral valve does to the left. Except for the number of its leaflets, its anatomic construction is similar, that is, the edges are joined to the delicate chordæ tendineæ which limit its motions; hence it is subject to the same ailments as its fellows, but for some unknown cause is less often visited by these afflictions. The *tempo* of the murmurs which arise in connection with disorders of this valve will coincide in all respects with those of the mitral. Hence tricuspid stenosis will be presystolic (auricular-systolic), and tricuspid insufficiency will be accompanied by a regurgitation which takes place during right ventricular systole (v. s.)

Tricuspid regurgitation seldom exists as an independent disease, except as the result of a congenital defect. It is prone to be one of the later complications of mitral disease.

The stenosis is still rarer than the incompetency.

Tricuspid murmurs are quite distinct and superficial, almost never heard higher than the third rib, hence are not easily overlooked, nor are they apt to be misinterpreted.

AORTIC VALVE MURMURS.

The outflow of blood through the aortic semilunar or sigmoid valve takes place during the systole of the ventricles. Alterations occurring at this orifice of such a nature as to offer an impediment to the outward current, such as narrowing of the passage, give rise

to a murmur which is synchronous with the apex beat and follows the first sound of the heart as a prolongation of that sound. The murmur will then be ventricular-systolic (v. s.), and the character of the obstruction will be stenosis.

If, by reason of enlargement of the aortic ring or owing to defects already expounded, the valves are unable to prevent the reflux of the current which closes them back into the ventricular chamber, then this backward flow or regurgitation of blood will also engender a murmur which succeeds immediately upon the ineffectual closure of the valves, that is to say during the period of diastole of the ventricles (v. d.) The murmur so produced is usualy a long-drawn, soft souffle which, as an index of disease, is the most trustworthy of all the adventitious heart sounds. Such a sound may replace or obscure the second aortic heart sound, since in some cases the stiffened segments make little or no efforts at closure, and the backward flow *begins* with the *beginning of diastole.*

A consideration of what is taking place in the cardiac chambers at this time will enable us better to understand the mechanism of this murmur. While the semilunar valves are closing, the ventricles are dilating with the in-take of blood from the auricles, which act continues not only during the time when the second sound is produced, but subsequently thereto until these chambers are filled, when another systole empties them; hence the murmur ceases *before* systole.

In many instances the defect of the aortic valve produces a double murmur, a combination of the two conditions described (v. s.+v. d.). Their relation in point of precedence will be described later.

THE PULMONARY VALVE MURMURS.

Acquired lesions of the pulmonary valve are pathologic curiosities. Even congenital lesions are rare. Murmurs are often heard in the pulmonary region, but most of these are either false or functional murmurs, or are transmitted from other seats, the areas being somewhat anomolous. Attention is directed to this condition of affairs in speaking of mitral affections.

However, two lesions of the pulmonary leaflets are recognized, stenosis and insufficiency. The first should give rise to a murmur synchronous with the emptying of the right ventricle, which is therefore ventricular systolic (v. s.); and the second, when manifested by a definite murmur, would be in time ventricular-diastolic (v. d.), but

these are perhaps the rarest of all the cardiac murmurs and the physician should be wary of asserting on insufficient evidence the presence of the lesion which seemingly gives rise to such a murmur.

FALSE MURMURS.

The name of *cardio-pulmonary murmurs* is given to false murmurs which closely resemble the sounds produced by organic disease of the valves, but which in reality are produced outside the heart as the result of disease within the thorax, or of neighboring organs.

The chief causes of such extra-cardiac murmurs are pressure exerted upon the heart, or displacement of the organ, as the result of disease of the pleura or the lung, effusion into the pericardiac sac, abdominal disease causing upward pressure, changes in the pericardium or in the lappet of lung which overlaps the heart.

Cavity contraction of the left lung with upward displacement is described under tuberculosis. This displacement gives rise occasionally to a systolic murmur heard at the second or third interspace, which may be mistaken for mitral regurgitation or even aortic disease. The displacement and the rareness with which valvular diseases are found in pulmonary tuberculosis should correct the error.

In a like manner a murmur is produced by disease of the left pleura. The sequelæ of this disease are thickening of the membrane, partial collapse of the lower lobe of the lung and contraction of the chest wall. The murmur is systolic, caused by the heart's impact displacing the air from the larger bronchi. This murmur is heard in the axilla, around to the left scapular angle as well as at the apex *and in the mouth*. The latter quality, in conjunction with the history, aids in diagnosis from organic mitral murmurs which are never heard in the mouth or over the trachea. Abdominal pressure, as in enlargements of the liver, and abdominal dropsies, have caused systolic murmurs which disappear after paracentesis.

Pleural effusions, accompanied by displacement of the heart, especially left-sided effusions, may be accompanied by a loud systolic murmur heard at both base and apex. It disappears after removal of the fluid. Osler and other writers call attention to the frequency of a systolic murmur heard about the cardiac apex in tuberculous subjects. The murmur resembles mitral regurgitation and is caused by the displacement of the air in the partially consolidated lappet, by the cardiac impact, as in the case of pleurisy. It is always more

distinct during expiration, and disappears when the breath is held after a full inspiration or when the patient lies down. Heard at the apex and in the left axilla, yet its most common seat and point of greatest intensity is in the second left intercostal space about two inches from the sternum. In doubtful cases of tuberculosis the presence of this murmur at the point indicated is strong evidence of consolidation. (See Pulmonary Tuberculosis.)

Changes in the pericardium resulting from previous inflammation are frequent causes of murmurs which are heard over an exceedingly limited area, viz., the sixth left interspace and over the seventh rib close to the ensiform cartilage, where the right ventricle is in contact with the chest wall. The sound is very superficial, short, sharp, systolic, never rough or blowing. The sound often resembles a reduplication of the first heart sound. It may disappear when the patient lies down, but not invariably.

False murmurs of this class, cardio-respiratory, often center around the pulmonary valve. In time they are systolic and are frequently to be heard in thin-chested individuals and children. They are best heard during forced expiration with the subject recumbent. They are present after great exertion or excitement and during convalescence from acute diseases.

The pulmonary area is a favorite seat for the anæmic murmurs.

Lastly emphysema, when it causes downward displacement, is frequently accountable for a murmur which is at one with the above described pericardial murmur in time, quality and location.

It will be seen, therefore, that the mere presence of a murmur, as has been said previously, by no means is indicative of organic valvular disease. Neither is the inability to discover such a murmur positive grounds for its exclusion. The diagnosis, therefore, should be based on *the ensemble of physical signs and especially on the consecutive changes* which the disability produces in the heart, sooner or later, in almost all cases of grave disease. Attention to these and the following points will usually lead to a correct interpretation of the conditions.

W. Gilman Thompson says that false and functional murmurs are always systolic, never diastolic. While we are not prepared to agree fully with this statement, nevertheless it must be admitted that the vast majority of them is systolic.

Osler lays down the following rules:—

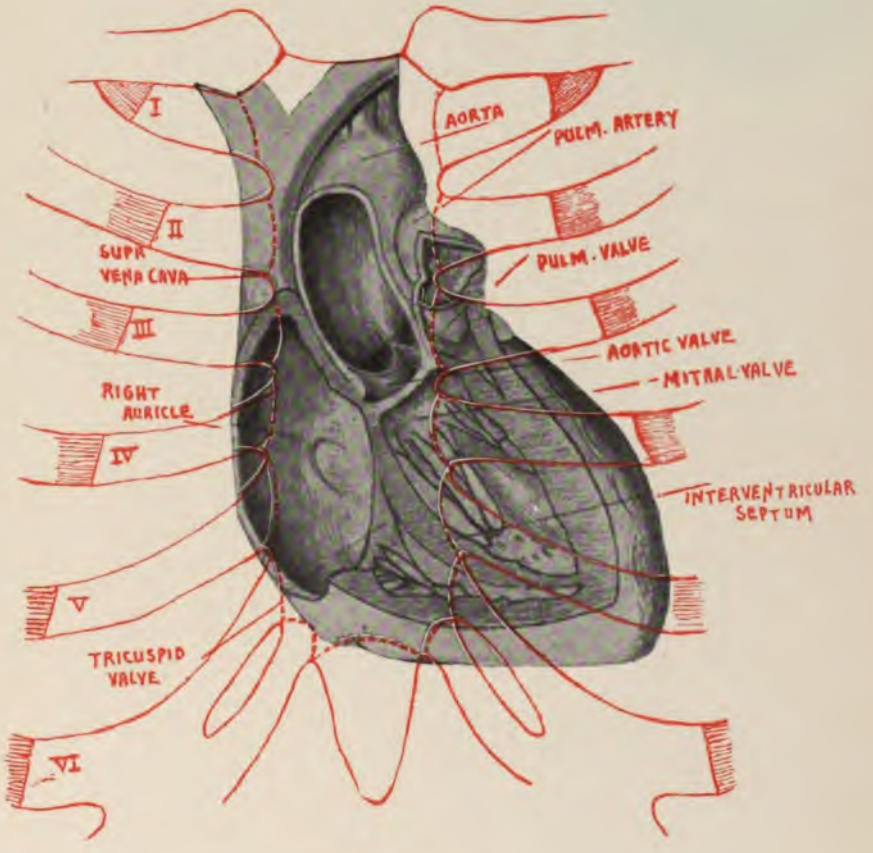


PLATE XII.

Relation of the Heart, its vessels and valves, to the Sternum and Cartilages.
Note relationship of Aortic and Mitral Valves.

From Cunningham's Anatomy
by permission of Wm. Wood & Co.

(1) In thin, nervous children a systolic murmur of soft quality is extremely common at the base, particularly at the second left costal cartilage, and is probably of no moment.

(2) A systolic murmur of maximum intensity at the apex, and heard also along the left sternal margin, is not uncommon in anæmic, enfeebled states, and does not necessarily indicate either endocarditis or insufficiency.

(3) A murmur of maximum intensity at the apex, with rough quality, and transmitted to the axilla or angle of the scapula, indicates an organic lesion of the mitral valve, and is usually associated with signs of enlargement of the heart.

(4) When in doubt it is much safer to trust to the evidence of the eye and hand than to that of the ear. If the apex beat is in the normal position, and the area of dullness not increased vertically or to the right of the sternum, there is probably no serious valvular lesion.

Organic Murmurs.	*False Murmurs.*
1. Quality oftenest blowing.	1. Often superficial, quality frictional.
2. Oftenest exactly synchronous with systole or diastole.	2. Time not synchronous with beginning systole or diastole. May precede or follow. Are rarely diastolic.
3. Definite seat of maximum intensity marked by anatomic point. Areas of propagation definite.	3. Seat, point of maximum intensity varies. Often heard at both base and apex without change in quality. Area of propagation irregular, not coincident with corresponding organic area.
4. Little affected by change in position or by holding breath. Intensity increased by forced expiration. Some heard only in recumbency.	4. Many disappear when patient is recumbent or upon sustained full inspiration.
5. If heard in mouth or trachea is aortic stenosis or aortic aneurysm. Mitral murmurs never.	5. Many heard in mouth, especially "air impact" murmurs.
6. Pulmonary engorgement common.	6. Pulmonary engorgement absent. May be anæmia.

SECTION X.

DISEASES OF THE HEART.

PERICARDITIS.

Definition. Pericarditis is an inflammatory process attacking the serous covering of the heart and its reflection upon the inner surface of the pericardial sac. A primary or idiopathic form is recognized, but is rare. Traumatic pericarditis may occur. Secondary forms arise in connection with rheumatism, tonsillitis, septic and tuberculous infections, scarlet fever and occasionally after other eruptive fevers and in the course of gout and Bright's disease. It occurs by extension in cases of inflammation of contiguous structures, especially of the heart, arteries and bronchial glands. Pleurisy, morbid growths, suppuration and occasionally other rarer causes are accountable for its presence.

Forms.—The disease may be divided conveniently into three forms:

1. Plastic or dry pericarditis.
2. Pericardial effusion.
3. Chronic or adhesive pericarditis.

The plastic or dry form may constitute the first stage of the succeeding varieties, but one-half of the cases do not advance beyond the dry form.

Plastic pericarditis is characterized by the small amount of fluid exudate, an amount too small to be recognized by physical signs. When caused by tuberculosis it may result in progressive chronic thickening without effusion.

Inspection gives no signs. Palpation may reveal a distinct friction fremitus, best-marked over the central and most superficial parts of the heart. The sign is oftener absent than present.

Auscultation reveals the friction sound, one of the most important, as well as one of the most distinctive, of physical signs. The sound originates from the rubbing together of the two roughened sur-

faces of the pericardium, hence is a superficial to-and-fro sound heard directly under the ear. Although caused by the systole and diastole of the heart, the rubbing is not perfectly synchronous therewith, being usually more prolonged than these sounds. It may be single, or, on the other hand, the rhythm may be multiple or galloping. Galloping rhythm is, however, more characteristic of the adherent form.

The sound is rough, grating, rubbing, creaking, or it may be loud and rasping. The creaking character has been likened by the French to that of new leather and is termed, *bruit de cuir neuf*. It is never blowing; never conducted in the direction of the blood currents; never heard over wide areas. It may be intensified by bending the patient forward. It is influenced by respiration and changes of position.

In some cases the sound is best heard over the apex or over the base, instead of in the area given.

Pericardial friction is not a phenomenon of long duration and disappears as soon as liquid accumulates within the sac.

Diagnosis. The only probable mistake which is apt to occur in the diagnosis of dry pericarditis is to confuse a pleuro-pericardial friction sound with a pure pericardial friction. The former is common in phthisis and pleuro-pneumonia. Time will usually resolve the difficulty since the pure pericardial sounds disappear after a brief interval, while the others may remain for protracted periods. Pleuro-pericardial sounds are not as a rule located over the anterior and superficial parts of the heart. Being caused by roughening in the left ventricular region, the sounds are confined to the left *border* of that organ.

Holding the breath does not affect the pericardial sound, but often causes a cessation of the pleuro-pericardial, particularly holding the breath after a deep inspiration. The respiratory rhythm further affects the last named by increasing its intensity during expiration and diminishing it with inspiration. It is less superficial and not apt to be a to-and-fro sound.

Double aortic murmurs are propagated into the neck, are permanent, not affected by respiration or position and are accompanied by a wholly different train of symptoms, which should prevent confusion.

PERICARDIAL EFFUSIONS.

The signs vary with the amount of the effusion. If considerable, *inspection* will show præcordial bulging, especially in children, widen-

ing and obliteration of the interspaces and a wavering, undulating impulse. In smaller effusions the impulse is tumultuous. As the amount of fluid increases the heart recedes from the chest wall and the impulse becomes invisible.

The veins of the neck show distention. There is often a prominence in the epigastrium due to the downward displacement of the liver. Œdema of the chest wall may be present.

Fig. 43—Showing area of dullness in pericardial effusion.

Mensuration. The expansion of the left lung is diminished, the left lateral circumference is increased.

Palpation. The apex beat gradually becomes less and less distinct and disappears. After it can no longer be felt in the fifth interspace it still may be palpable in the fourth. Friction fremitus has now disappeared over the right ventricle, but may persist longer over the base of the heart. Fluctuation can very rarely be detected.

Percussion furnishes the most striking symptoms of effusion. As the fluid accumulates the area of cardiac dullness enlarges in all directions. The edges of the lungs are pushed aside, even the diaphragm

and liver are depressed. The area is now rudely triangular or pear-shaped with its base across the sixth or even the seventh interspace, its apex or stem pointing to the inner end of the left clavicle. In very large effusions dullness may extend from nipple to nipple, which is never the case in cardiac hypertrophy. This dullness is rendered the more striking and abrupt by the hyper-resonance of the neighboring compressed lung.

Fig. 44—Effusion into pericardial sac, shading insensibly into the diaphragm.

Rotch's sign of effusion is dullness in the fifth interspace on the right side. Ewart's sign is dullness or impaired resonance posteriorly above the angle of the left scapula. Both are valuable. Pressure upon the left lung may impair the pulmonary percussion note, rendering it flat or vesiculo-tympanitic (Skoda's resonance). The change is most marked in the left axillary region as high up as the nipple. It is altered by changes in the position of the patient, as is also the area of dullness at the scapular angle; both may disappear when the patient leans forward.

Auscultation. With the increase in the effusion the heart sounds

become gradually weakened and muffled and finally are almost inaudible. Friction sounds may persist over the base of the heart after they have disappeared from over the ventricular area, or may be heard when the patient is erect, disappearing when he lies down. Generally the heart sounds may be heard at the top of the sternum after they have become inaudible over the præcordia.

The heart's action is increased and irregular. A systolic murmur may be present and give rise to the erroneous diagnosis of mitral valve disease, and the pulmonary second sound may be manifestly accentuated. The breathing sound referred to above, heard in the axillary region, is feeble or even tubular in case of sufficient pressure. The quality changes somewhat with variations in the position of the patient.

Diagnosis. The diagnosis presents many difficulties, in spite of the well-marked signs set down. The disease is often overlooked and even unsuspected when the sac is filled to repletion with fluid. In cases of rheumatism, especially in children, the heart should be examined daily.

The condition which most closely resembles effusion is dilatation of the heart. The following are the principal points of difference:

Dilatation.	*Effusion.*
Inspection. Impulse visible except in stout persons. Wavy.	Impulse invisible.
Palpation. Impulse felt. Unchanged by forward inclination of patient.	Impulse not felt when erect. May be palpable when patient leans forward.
Percussion. Dull area not triangular, increases downward and to left in mitral stenosis, in other forms it never reaches so high on left of sternum.	Dull in fifth and even sixth interspaces on both sides; is triangular-shaped, reaches high as second rib on left of sternum; upper limit may shift with change of position.
Auscultation. Heart sounds valvular and clear, especially second sound.	Sounds distant and muffled.
Dyspnœa less urgent and influenced by exertion.	Dyspnœa not so influenced
Slow development, no pain, no fever.	Rapid development, pain, fever.

The most striking point of difference is that dilatation never reaches a degree in which the dull area simulates that of effusion, *without the presence of visible pulsation* and never produces Skodaic resonance in the axillary region.

CHRONIC PERICARDITIS; ADHESIVE PERICARDITIS.

The signs are most pronounced in children, in whom the condition is prone to remain as a memento of rheumatism. The disease ordinarily leads to dilatation and hypertrophy with insufficiency of an extreme grade, and the physical signs ultimately become the signs of that affection. Other manifestations vary exceedingly.

Inspection. The præcordia in the post-effusion stage is retracted, especially in young subjects: later, bulging and asymmetry succeed owing to the cardiac enlargement. The impulse is diffuse, multiple or wavy, increased in area, often extending from the third to the sixth rib and beyond the nipple. It may be displaced in any direction, especially downward or to the right. Each systole may produce a retraction of the chest wall, a valuable sign of adherence when present. Adhesion to the diaphragm gives rise to a systolic tugging at the points of attachment, particularly at the seventh and eighth ribs in the left antero-lateral region, and behind between the *eleventh and twelfth ribs on the left side*. This visible retraction of the chest wall behind with each systole is known as Broadbent's sign. In such attachments the diaphragm does not descend with inspiration, hence the natural respiratory excursion of the epigastrium is absent. The same restriction of diaphragmatic movement occurs, however, in pleuritic effusions and emphysema *above*, and in ascites, abscess or tumor *beneath* the structure; hence the sign must not be interpreted as invariably indicating adhesions, as is sometimes stated. The diastolic collapse of the cervical veins due to their sudden emptying, with expansion of the chest wall, is known as Friedreich's sign. Osler says its significance is of little moment.

An important sign related to adherent pericardium, but by no means confined to that disease, is known as Litten's sign, described by him in 1892 and denominated "The Diaphragm Phenomenon." It is a visible, undulating, ascending and descending wave or shadow to be seen with respiration between the sixth intercostal space and the free border of the ribs, crossing the latter at an acute angle. It is produced by a stripping off of the diaphragm from the chest wall,

and is synchronous with the movements of the midriff. It is most apparent in the axillary or parasternal line when viewed in a strong light. With the patient recumbent, the arms extended above the head, he is directed to take a long breath, which must be abdominal and not costal. Litten also states that by laying the patient on his belly the wave is sometimes seen on deep breathing posteriorly in the lower thoracic zone. It may be seen in a good artificial light and may sometimes be noticed with the patient standing or sitting, as well as when lying down. The sign is absent in very young children and changes its character in old age owing to the calcification of the cartilages. It is a common phenomenon of health and is present in over ninety per cent. of individuals, in fact in all cases where the diaphragm ascends and descends normally with respiration.

The normal shadow is about three inches in extent (6-7 cm.—Litten) and is more marked in men than in women and children. When equal and unbroken on both sides it indicates normal pulmonic conditions or healthy lungs.

It is exaggerated in both health and disease when the respirations are uniformly increased. Exaggeration on one side means vicarious respiration on that side and diminished function on the opposite. It is diminished on both sides equally in asthma, emphysema and other conditions which diminish uniformly the respiration volume. In such cases Litten says the sign is more apparent posteriorly than anteriorly. It is diminished on one side in the unilateral affections of the lung or pleura, especially unilateral tuberculosis, of which it is an early and reliable sign, in pleurisy, in hypertrophy of the heart and in pericardial effusions. Pleural and pericardial adhesions break the wave in a variety of ways. When the adhesions are extensive the wave is absent. It is absent in advanced tuberculosis and other diseases accompanied by considerable consolidation, and in cases of collection of air or fluids within the pleural sac.

Accumulations of fluid, tumors below the diaphragm, and advanced pregnancy mechanically inhibit the shadow, and thus its absence serves to locate them. Litten mentions that it is present in subphrenic abscess.

Palpation. The diastolic shock or rebound of the heart is felt when the hand is placed over the præcordia. This is regarded as the most positive sign of adherent pericardium. Systole may cause a retraction of the chest wall, to be followed by the shock. The apex

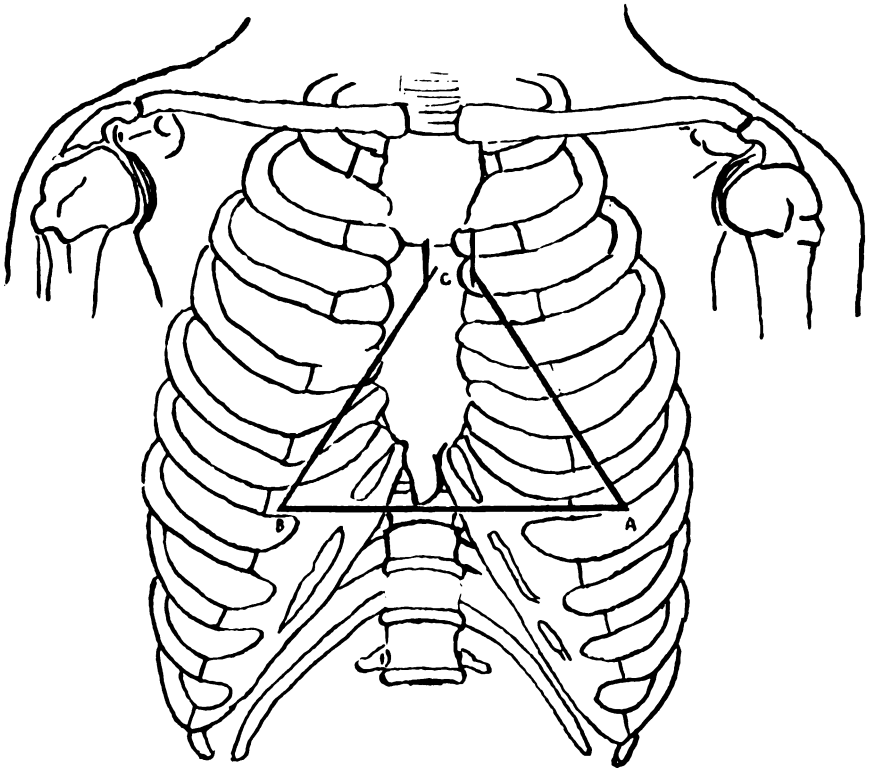


PLATE XIII.

A B C The Area of Dullness in Pericardial Effusion as shown by the X-Ray Examination. Diaphragm pushed down. Heart Apex at A.

position does not change to the left as in the normal heart, when the patient is turned on the left side, owing to the adhesions.

Percussion. The area of cardiac dullness is increased upward and to the left, reaching in extreme cases the first interspace. Adherence between the pericardium and the pleura may prevent the left and upper margin of the dull area from changing with deep breathing, as pointed out by Williams. Osler finds that considerable adhesion of pleura to pericardium, or of pleura to chest wall, none the less allows almost unrestricted motion.

Auscultation. The signs may be negative. At best they are uncertain. The heart rhythm may be galloping or fœtal. A systolic or presystolic murmur is often present, heard over the apex. The signs of dilatation and insufficiency ultimately predominate when that condition becomes paramount.

Auxiliary signs. That pericarditis is a painful disease is a widespread error. Pain is severe in a few cases, in others it is absent or only slight. It may be referred to the xiphoid cartilage. Fever is generally present but is not high. In intercurrent cases it augments the existing temperature. In rheumatism hyperpyrexia may occur. Dyspnœa of slight grade occurs in the plastic form; with effusion it is generally sufficient to draw attention to the heart.

The pulse is rapid and small. In adherent cases the *pulsus paradoxus* of Kussmaul is a symptom of note. It consists of an increase in frequency with gradual weakening and even disappearance of the pulse during inspiration. Initial chill is occasional.

HYDROPERICARDIUM.

During the course of those diseases which produce general anasarca or dropsy, fluid may accumulate within the pericardium in considerable quantity. The disease is then known as hydropericardium. The condition is unaccompanied by inflammation of the sac, but occurs in the course of such chronic diseases as nephritis; the valvular heart affections, cirrhosis of the liver, cancer, and tuberculosis. The effusion is sometimes stained red. Pleural effusions may be complicated by transudate into the sac, in which case one or the other effusion is very apt to be overlooked. Occasionally the fluid collects after scarlet fever.

The physical signs are those given under pericarditis with effusion. The malady is insidious and, occurring in the course of diseases accom-

panied by dyspnœa causes few additional signs, hence should be held in remembrance.

The normal state of the pericardium during life is probably like that of the freshly-opened peritoneum, that is, humid but never wet, hence the small but varying amount of fluid found in the pericardial sac after death is probably a post-mortem transudate.

Hæmopericardium, or blood in the pericardial sac, is due generally to the rupture of an aneurysm into the sac. Aneurysms of the first part of the aorta frequently so rupture owing to the attachment of the sac being about two inches above the origin of the great vessels (Gray). The aneurysm may be of the coronary arteries or of the heart wall. Rupture or wounds of the heart are less frequent but possible causes. Death quickly intervenes in all these cases with signs of heart failure and dyspnœa. Percussion shows the characteristic dull area.

Pneumopericardium. Gas in the pericardial sac is a rare condition, but may result from a tuberculous perforation from the lung or the œsophagus, or perforation through the diaphragm from cancer of the stomach. It has followed stab-wounds.

Pericarditis with effusion quickly follows and the admixture of gas and fluid gives rise to a movable area of percussion dullness associated with tympany.

On auscultation friction sounds are heard admixed with splashing, bubbling and churning sounds which are remarkable but characteristic. The heart sounds are obscured and, if heard, are feeble and distant. Death is not long delayed.

PHYSICAL SIGNS AND SYMPTOMS OF THE VARIOUS FORMS OF VALVULAR DISEASES—MITRAL INSUFFICIENCY.

This is the most frequent form of valvular disease, yet it may exist for years without the patient being aware of any defect. The first manifestation is shortness of breath on going up hill, up-stairs or following unusual exertion.

As explained in the consideration of valvular defects, the ineffectual closure of the mitral valve allows a certain amount of blood to be thrown back into the auricle with each systole. The auricle is receiving at the same time the blood from the lungs. The two currents cause an excessive diastole, which inevitably results in dilatation. Owing to the excess amount of work necessary to expel the augmented quantity

of blood, auricular hypertrophy supervenes. As soon as this condition is reached each auricular systole forces an increased quantity of blood into the left ventricle, with the same results as in the case of the auricle, viz.—first dilation, then hypertrophy of that part.

The resultant action of mitral incompetency upon the right heart is explained as follows:

The over-filled left auricle impedes the emptying of the pulmonary veins into the cavity, as occurs in normal hearts during diastole. This venous engorgement and the consequent increased interpulmonary pressure prevents the free expulsion of blood from the right ventricle into the pulmonary arteries, and dilation of that chamber results, with subsequent hypertrophy. This hypertrophy causes an accentuation of the pulmonary second sound. The right ventricular dilation results in insufficiency of the auricular-ventricular valves on that side and finally the right auricle is involved. Venous pulsation in the neck indicates its occurrence, and betokens venous engorgement elsewhere. The stomach, omentum, kidneys and liver are especially involved. General dropsy supervenes; anasarca is accompanied by albuminuria. The association of liver pulsations with tricuspid disease has been pointed out and occurs here as a late lesion. Owing to the pulmonary engorgement haemorrhage is apt to occur and is not confined to the late stages of the disease. In mitral insufficiency it may be an early symptom.

Attacks of bronchitis are frequent, due to the same predisposing cause.

Symptoms. The disease naturally divides itself into two periods, during which the symptoms and physical signs vary greatly. These are the period during which compensation is perfect, and the period when compensation is no longer maintained and during which the ever-increasing venous engorgement originates most of the evils of the disease. For convenience these periods will be referred to as the first and second stages of the malady.

Inspection. The area of the visible pulsation is increased and diffused, especially so in children. The apex impact is in the nipple line or beyond and displaced a little downward, appearing usually in the sixth interspace. In children the præcordia bulges. The face shows a certain amount of venous congestion especially over the cheek bones where the veins are enlarged and thread-like. The lips and ears show a bluish tint: this in cases of long-standing, even with compensa-

tion. Clubbing of the fingers occurs and is pronounced in children.

In the first stage dyspnœa is not pronounced, but shortness of breath follows unusual exertion. With the second stage come marked cyanosis and a jaundice tint, and dyspnœa is profound. Irregularity of the heart's action, palpitation and diffusion indicate dilation. Epigastric pulsation occurs in the second stage, when the right heart participates.

Palpation. First stage. Impulse forcible and even heaving. Second stage. Impulse diffused, weak and irregular, often arythmic and wavy. Thrill is rare. If present it is felt at the seat of the apex beat and is synchronous with the first sound. The pulse is regular and full during compensation, but in the second stage becomes small, easily compressed and irregular. When once this irregularity sets in it seldom ever afterwards disappears.

Percussion. The heart boundaries are enlarged *laterally* much

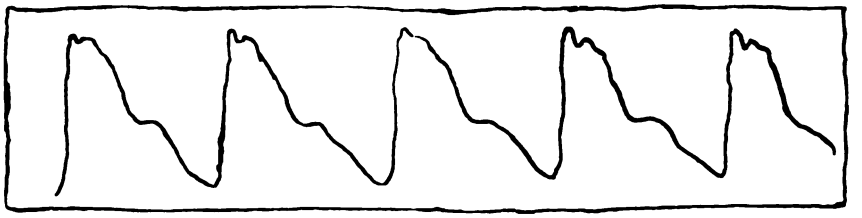


Fig. 45—Pulse tracing in a case of mitral regurgitation in a well compensated state (Patton).

more than *vertically*. This diagnostic point is referred to under pericarditis with effusion. When the right ventricle becomes involved this area increases on both sides of the sternum. No other form of valve disease so materially increases the transverse diameter, which may extend from a point an inch beyond the right sternal margin to the left anterior axillary line.

Auscultation. A systolic murmur is heard which has its greatest intensity at the apex. This murmur, as already stated, may follow the first heart sound as a prolongation thereof, or it may wholly replace the systolic sound. Its character is generally soft and blowing, but may be musical. Its strength or loudness depends greatly upon the ventricular contraction. Change in this respect in the downward scale portends evil. The word *soft* used in this connection must not be interpreted to mean *feeble*, but as being opposed to high-pitched and

shrill. This murmur is transmitted directly around the ribs into the axilla and can usually be heard as far as the angle of the scapula. Sometimes the murmur is audible in the recumbent posture, and disappears when the patient is erect. At the apex as a rule the second heart sound heard during diastole, is loud and decisive. It is due to pulmonary accentuation. In cases of great hypertrophy affecting both sides of the heart, a strong systolic blowing sound is the only murmur heard. In mitral regurgitation the disturbance in the heart's

Fig. 46—Mitral insufficiency, showing great increase in the transverse area. Apex beyond the anterior axillary line in 7th interspace. Some emphysema is associated. During fourteen years equilibrium has been well maintained, until within three months of present time.

rhythm is chiefly in the direction of shortening the intervals between the systoles.

The above describes the typical state of affairs, and cases are frequently encountered which will conform with exactness to the picture. The principal *variations* are that sometimes the murmur is best heard along the left border of the sternum, as pointed out by Naunyn, or at

the base of the heart where it may be loudest, and may be audible only in that situation, as mentioned by B. Foster. These instances are rare. Sometimes it is heard over the entire chest. There may be associated a presystolic mitral murmur. The association of this presystolic with the systolic mitral murmur is the only *absolute* ante-mortem evidence

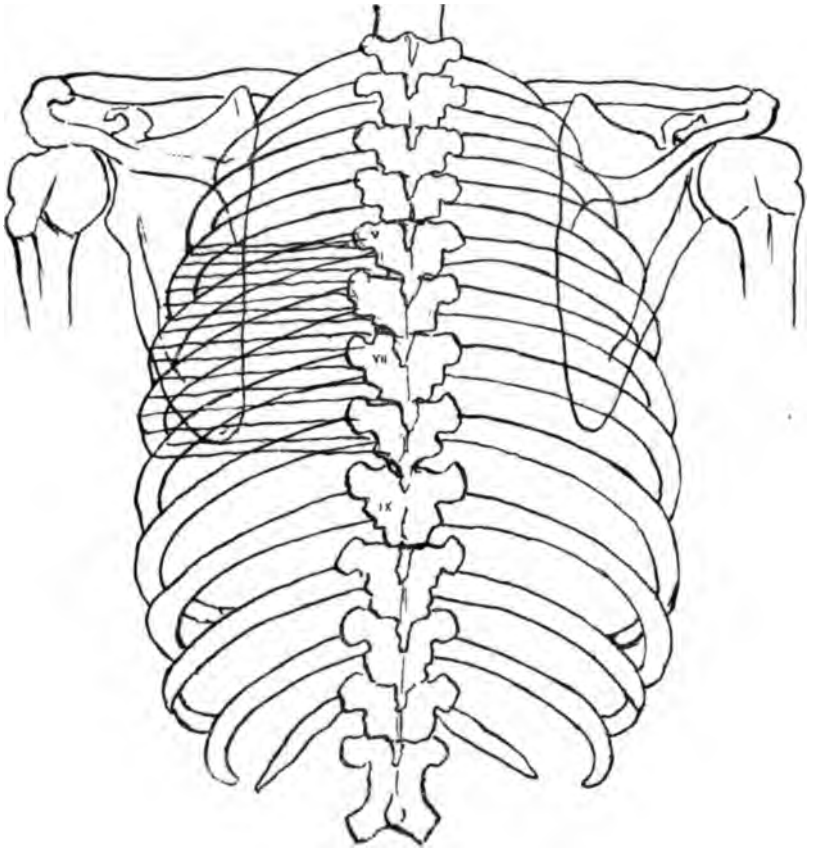


Fig. 47—Dorsal area of murmur, mitral regurgitation. Patient shown in photograph (Fig. 46).

which we possess of positive incompetency of the mitral valve, since dilatation of the ring with relative incompetency and sclerosis will induce an identical train of sequences. Late in the disease tricuspid regurgitation occurs and a soft, low systolic murmur in the tricuspid ensiform region indicates it. This latter complication is also evinced

by a marked accentuation of the pulmonary second sound of a clacking character, due to the increased tension in the pulmonic vessels.

Diagnosis. Four cardinal points are to be kept in mind:

Apex beat displaced to the left and downward.

Transverse area of dullness much increased.

A systolic murmur with greatest intensity at apex, carried back to angle of scapula with diminishing intensity.

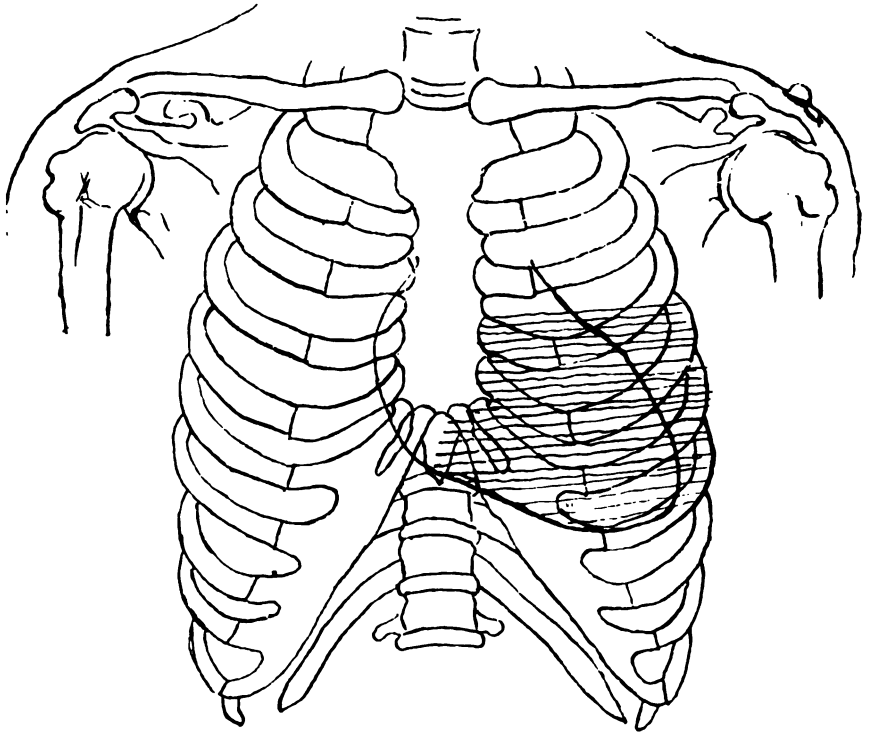


Fig. 48—Percussion outline and area over which is heard murmur of mitral insufficiency in patient whose photograph is shown (Fig. 46).

Accentuation of the pulmonary second sound.

The relative intensity of the two *second* sounds heard at the base of the heart, the pulmonic and the aortic, vary much in normal individuals, as pointed out by Vierordt. In children the pulmonic sound is almost invariably the stronger and louder. In old age the condition is exactly reversed. In adults the percentage of cases of pulmonic predominance diminishes with each decade.

MITRAL STENOSIS.

It has been questioned whether mitral stenosis can exist without some degree of regurgitation. Balfour maintains that it cannot.

The mechanism. The difficulty of expelling its blood through the narrowed orifice causes the left auricle to dilate and to hypertrophy. As long as it is equal to the extra task imposed upon it compensation is maintained, but as its systole is prolonged and rendered more difficult, the tension in the pulmonary veins increases by reason of the impedi-

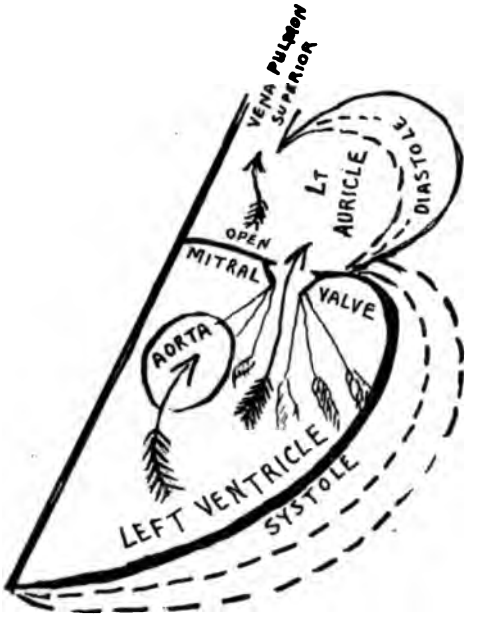


Fig. 49—Diagram illustrating mitral insufficiency.

ment to their outflow, and the right ventricle is thus forced to increase its work in order to overcome the augmented pulmonary tension against which it is pumping. Venous congestion of the systemic veins indicates that the right heart is no longer capable of equalizing the circulation. Competency is not maintained for the protracted periods often observed in the foregoing variety of the disease.

The heart of mitral stenosis is in marked contrast with the incompetent heart. It rarely attains any great size, and the increase is principally in the left auricle and the right ventricle, which latter forms the greater part of the lower anterior cardiac segment.

Symptoms. Cardiac palpitation, dyspnœa and stitch-like pains about the apex are frequent symptoms. The patients are prone to be anæmic and attacks of recurrent endocarditis are apt to occur. They are indicated by febrile attacks and marked aggravation of the circulatory symptoms. As compensation fails venous engorgement of the lungs results and induces symptoms which differ in no wise from those described under insufficiency.

Inspection. As the left ventricle is not enlarged there is no bulging in the apex region, but in children there may be a prominence

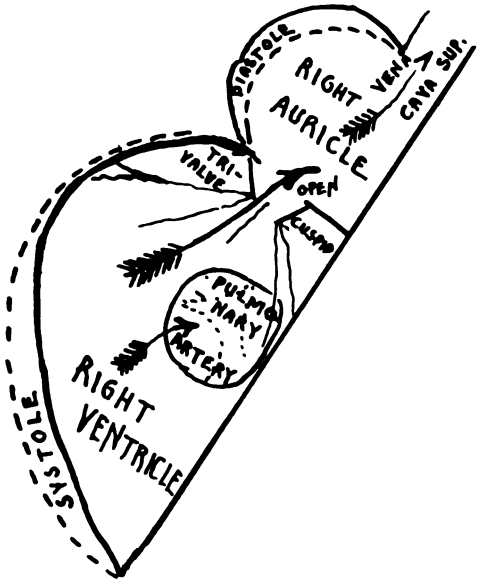


Fig. 50—Diagram illustrating tricuspid incompetency.

of the ensiform and lower sternal regions, or of the left lower costal cartilages caused by the hypertrophy of the right ventricle. The apex beat is pushed but little to the left, seldom beyond the nipple line. Often it is not well-defined, but is replaced by a diffused pulsation to the right of the apex beat in the region of the fifth and sixth left costal cartilages, which is due to the impact of the hypertrophied right ventricle, which, as already explained, lifts the apex from the chest wall. The pulsation seen in the third and fourth left interspaces in persons with thin chest walls has been generally ascribed to the enlarged left auricle. This is very probably erroneous, the pul-

sation being in reality due to the impulse of the conus arteriosus of the right ventricle.

Engorgement and pulsation of the jugulars indicate failure of the right ventricle to maintain equilibrium.

Palpation. The heart impact is diffused and is felt most forcibly in the region to the right of the lower sternum. Epigastric pulsation is not rare. Palpation furnishes the most definite, significant and trustworthy sign of mitral stenosis. It is the sharp thrill which is felt over a very limited area whose center is the fourth interspace just within the nipple line. It may extend as high as the third or as low as the fifth. It is rough and rasping in quality, begins during diastole as a purring fremitus, gathers force with the auricular systole, which is its true cause, and terminates as an abrupt shock with the beginning of ventricular systole. The sign is pathognomonic. When

Fig. 51—Pulse tracing in a case of mitral stenosis (Patton).

not appreciable during repose it may be elicited by slight exertion. Anders states that turning the patient upon the left side and elevating the arms accomplishes the same result. When compensation fails the thrill disappears and the lesion may be overlooked. With reëstablishment of equilibrium the murmur returns.

Percussion. The increase in percussion dullness is principally to the right and may extend as far as two inches beyond the sternum. On the left it seldom extends beyond the nipple-line and is due for the most part to increase in the size of the right ventricle. The vertical boundaries are also increased and the base-line moves up to the second interspace.

Auscultation. A rough, vibrating thrill or purr is heard in the apex circle or a little within and above the apex point. In time it is diastolic and may occupy the entire diastolic period, or it may be present only during the latter part of diastole, that is to say during auricular systole. In either case it terminates abruptly with the ven-

tricular shock. Its time is best ascertained by combined palpation and auscultation. When it fills the entire diastolic period the two parts of the murmur may show differences in quality sufficient to divide it (Gairdner).

In the second left interspace the second sound is unmistakably

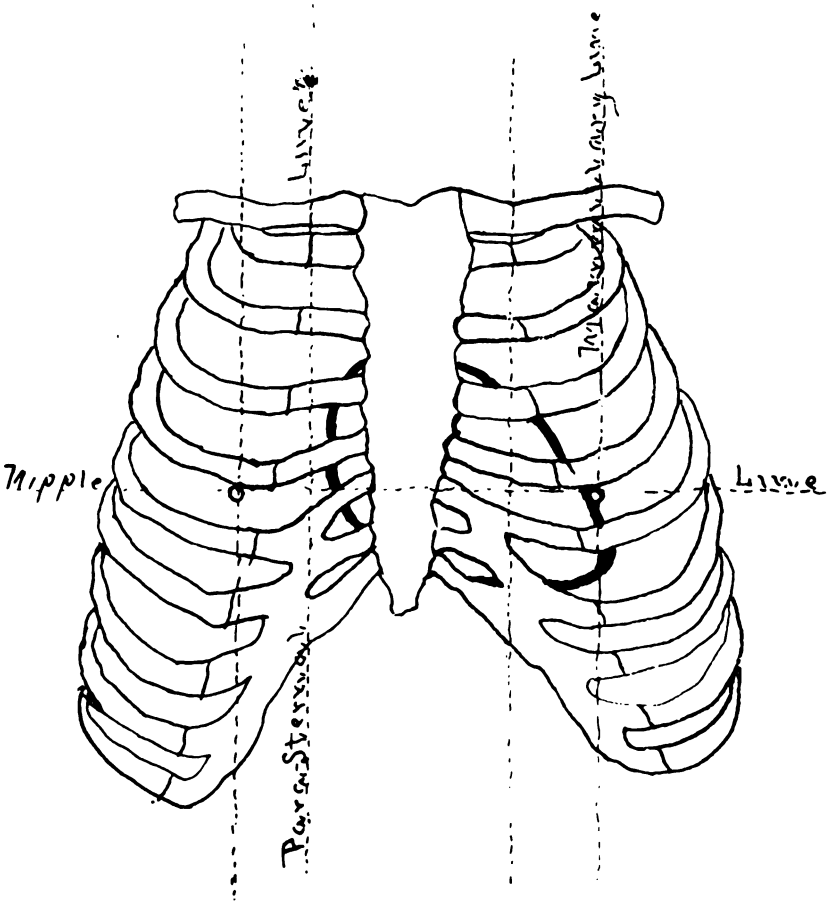


Fig. 52—Enlargement of heart in mitral lesions (Patton).

accentuated and often reduplicated. Sansom says this reduplication is present in one-third of the cases.

The first heart sound heard in the apex region is sharp, clear and short. Whether this sound arises from accentuation of the tricuspid closure or whether due to the sharp muscle contraction of abbreviated

systole, as held by Broadbent, remains an open question. Even when compensation has failed, the thrill disappeared and the cardiac rhythm has become galloping, this sound retains its clearness.

The regularity of the heart's action is affected as shown by the cardiograph. The diastolic intervals vary greatly in length and the intervals between the systoles are prolonged.

The murmur of mitral incompetency may be associated with the presystolic murmur, and later with that of tricuspid incompetency. Mitral stenosis is generally a primary affection.

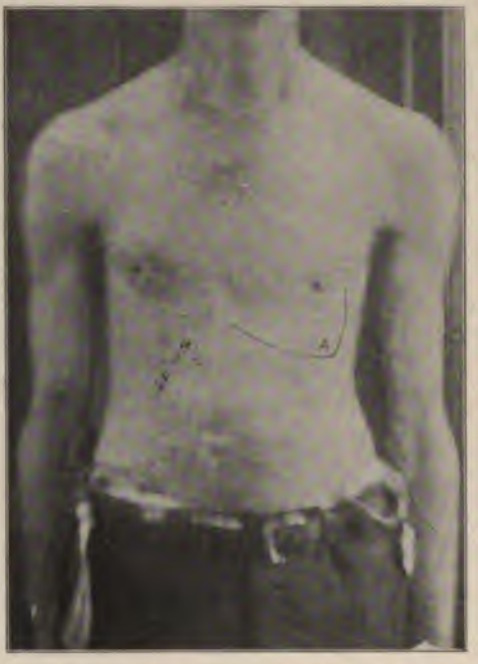


Fig. 53—Mitral insufficiency with large area of hypertrophy. Patient is laborer with powerful physique. Murmur limited to small area on back to which it is directly transmitted. See Fig. 54.

The Pulse. The radial pulse is small in volume, irregular and easily compressed, and as the power of the right ventricle diminishes these characteristics increase. So long as equilibrium is perfect, the pulse is small, and may be quite regular.

The terminal symptoms are the same as those of incompetency, except that dropsy is less apt to occur. Pressure of the enlarged auricle upon the left recurrent laryngeal nerve, causing paralysis of the vocal cords, is mentioned by Herrick and by Osler.

MITRAL INSUFFICIENCY AND STENOSIS.

The combined lesion is more common than a pure stenosis, as a study of the valve conditions would lead one to suppose. The physical signs of the two conditions are then combined. The hypertrophy, the thrill, the accentuation of the second sound, and a double murmur with seat of intensity in the mitral area, summarize the phenomena. The

Fig. 54—Small area on the back to which the mitral murmur (Fig. 53) was directly transmitted. It was very faint in the axilla. The figures are on the tips of the vertebral spines.

pulse in this case is small, irregular and frequent. The sequelæ are those of incompetency.

AORTIC INCOMPETENCY.

Incompetency of the aortic valve is the best-defined and the most easily recognized of valvular lesions.

The mechanism of the disease is as follows:

The insufficient semilunar valves permit a portion of the blood pumped into the aorta to regurgitate into the left ventricle. The regurgitation occurring at the time when that chamber is filling from its auricle, the effect of the double supply of blood, is to cause dilatation of the ventricle, which is ultimately followed by its hypertrophy. As the two processes act continuously the necessity for ever-increasing

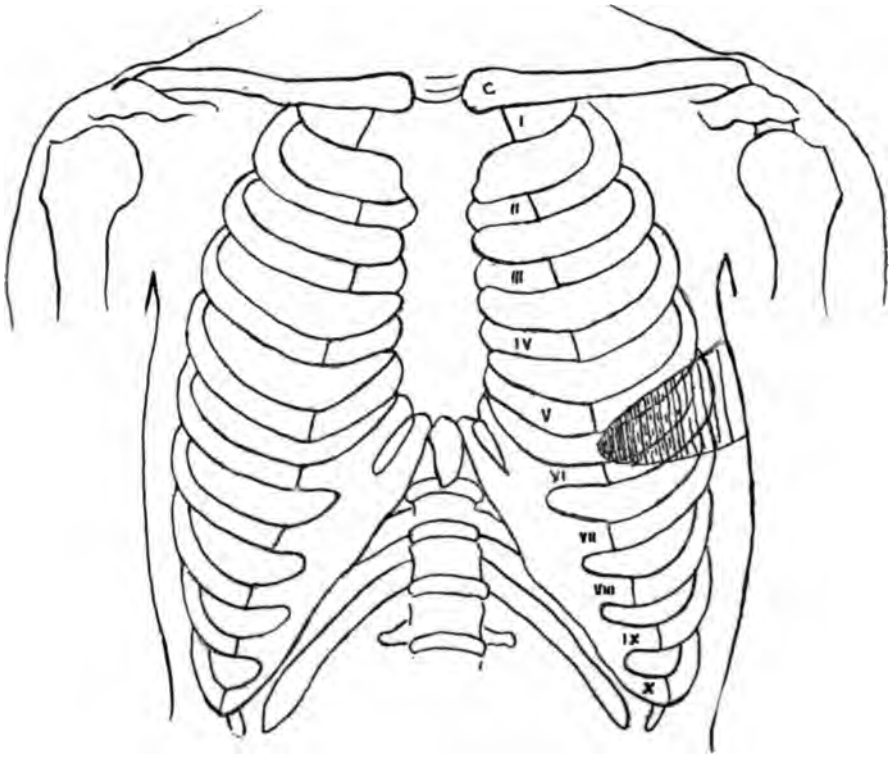


Fig. 55—Mitral systolic murmur—seat of greatest intensity and direction of propagation with diminishing intensity.

hypertrophy is manifest. The muscular endowment of the ventricle walls allows it to attain a degree of hypertrophy which surpasses that possible in the case of any other portion of the heart. Cases have been recorded in which a heart weight of forty-eight ounces was reached. As long as the two processes go on harmoniously equilibrium is maintained; no evil results follow, and the discovery of the condition may be accidental.

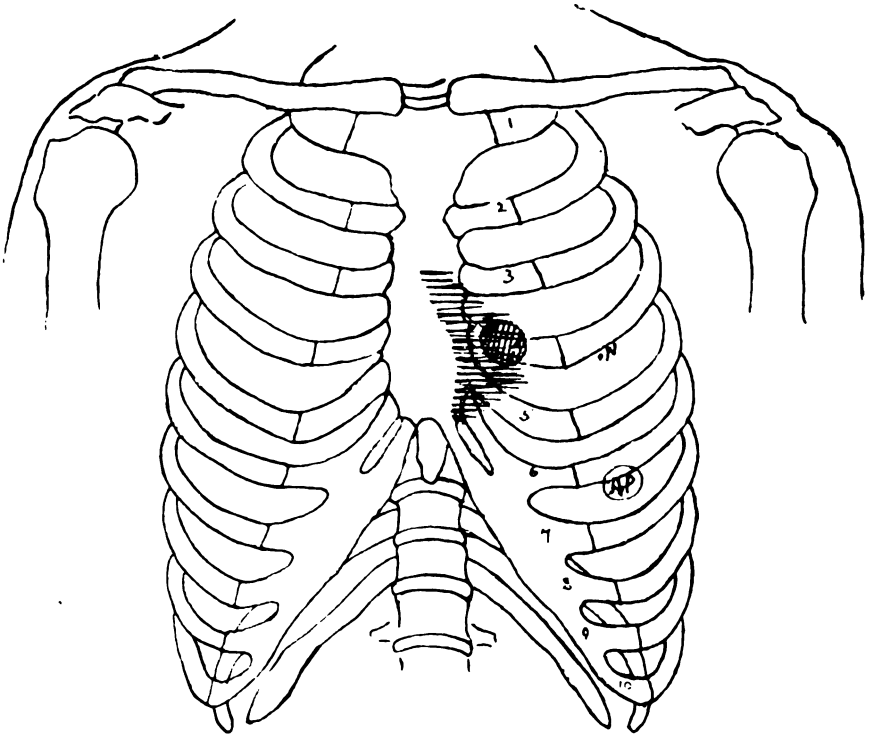


PLATE XIV.

Aortic Diastolic Murmur, seat of greatest intensity and direction of propagation. AP. Position of Apex.

The difficulty experienced by the left auricle in ridding itself of its contents, owing to the raised pressure in the ventricular chamber, causes it to dilate and then to hypertrophy, and to increase the length of its systole. The raised pressure within the veins of the lungs thus produced must be counter-balanced by increased pump-work on the part of the right ventricle, hence inevitably, after a longer or shorter time, the right heart shows secondary dilatation and hypertrophy, in which the dilatation is finally paramount. The student will understand that pathologic results are not always easily distinguished from

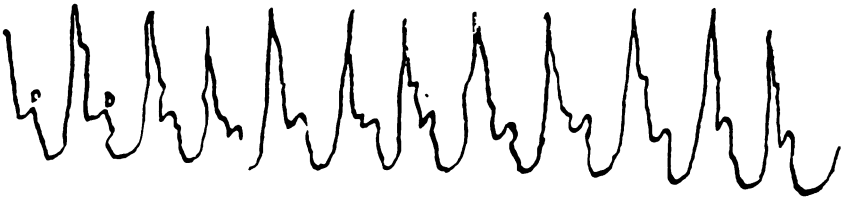


Fig. 56.—Pulse of aortic regurgitation. Apex acute. The height to which the lever rises is materially influenced by the amount of pressure made upon the button. D Aortic valve closure. Dicrotic notch marked. Predicrotic notch absent.

causes, and that the mechanism and order of sequence above given may be diversified on quite as reasonable grounds as those here assumed.

PHYSICAL SIGNS.

Inspection. The apex is seen in the sixth or seventh interspace, as far to the left as the anterior axillary line. The impulse is diffused and undulatory during the dilatation period, wide and forcible during hypertrophy. Bulging is seen in children; the entire præcordia is raised. Occasionally during systole there is a depression of the præcordia between the sternum and the nipple, due to atmospheric pressure.

Palpation. The character of the impact is wavy and undulatory, or strong and heaving.

Percussion. The area of dullness is enormous in advanced cases, attaining here its greatest limits. The transverse area extends to the left beyond the anterior axillary line and downward as far as the seventh interspace.

Auscultation. A murmur is heard with maximum intensity in the second right interspace (aortic cartilage). It is transmitted down-

ward along both borders of the sternum towards the apex and the ensiform cartilage.

Since it is produced by the reflux of blood during the filling of the ventricle the murmur occurs during diastole, and therefore *replaces the second sound of the heart*, or is intermingled therewith in cases where the leaflets are still capable of closure. In these cases both the murmur and the valve-sound may be distinct. The quality of the murmur varies greatly. Oftenest it is a long, soft, souffle, but may be harsh and rasping. Osler says it is the most trustworthy of all cardiac murmurs. We think it is not more indicative than the presystolic mitral thrill.

Associated Murmurs. In many cases of aortic incompetency the arch of the aorta has become roughened by atheromatous deposits. This roughening gives rise to a systolic murmur which is present in about one-third of all cases. The roughened semilunar valve segments also are wont to originate a murmur by the passage of out-bound blood over their surfaces. The presence of these murmurs is often interpreted to mean aortic stenosis, although only a small proportion of the cases show any real narrowing of the aortic orifice. Both sounds are systolic and may be heard beyond the aortic area. In most cases of aortic incompetency the ventricular dilatation and hypertrophy enlarge the mitral ring, causing relative insufficiency of its valves, giving rise to the systolic murmur of mitral incompetency. That this murmur arises at the mitral valve and is not a conducted murmur is shown by the fact that its quality is often materially different from the systolic murmur just described as arising from the aortic valve. This aortic murmur is rough, high-pitched; the mitral soft and blowing.

Flint Murmur. Yet another murmur is heard in the mitral or apical area, but is less common than the systolic bruit. This second murmur is known from its describer as the Flint murmur. Its quality is rumbling, its area limited and its time presystolic or diastolic. Whether it is due to the inflowing blood from the left auricle, or to the reflux current from the aorta producing vibration of the mitral leaflets, is uncertain. Flint believed that the dilatation of the ventricle prevented the mitral leaflets from folding completely backwards against the chamber walls and, thus acting as an impediment, produced the bruit.

The pulse is described elsewhere. The delayed radial pulse which follows the apex beat after a distinct interval is characteristic, and the

length of the interval increases with the increase of the incompetency. In the carotids, especially the right, the systolic murmur may be quite as apparent as at the cartilage, and the second aortic sound may be

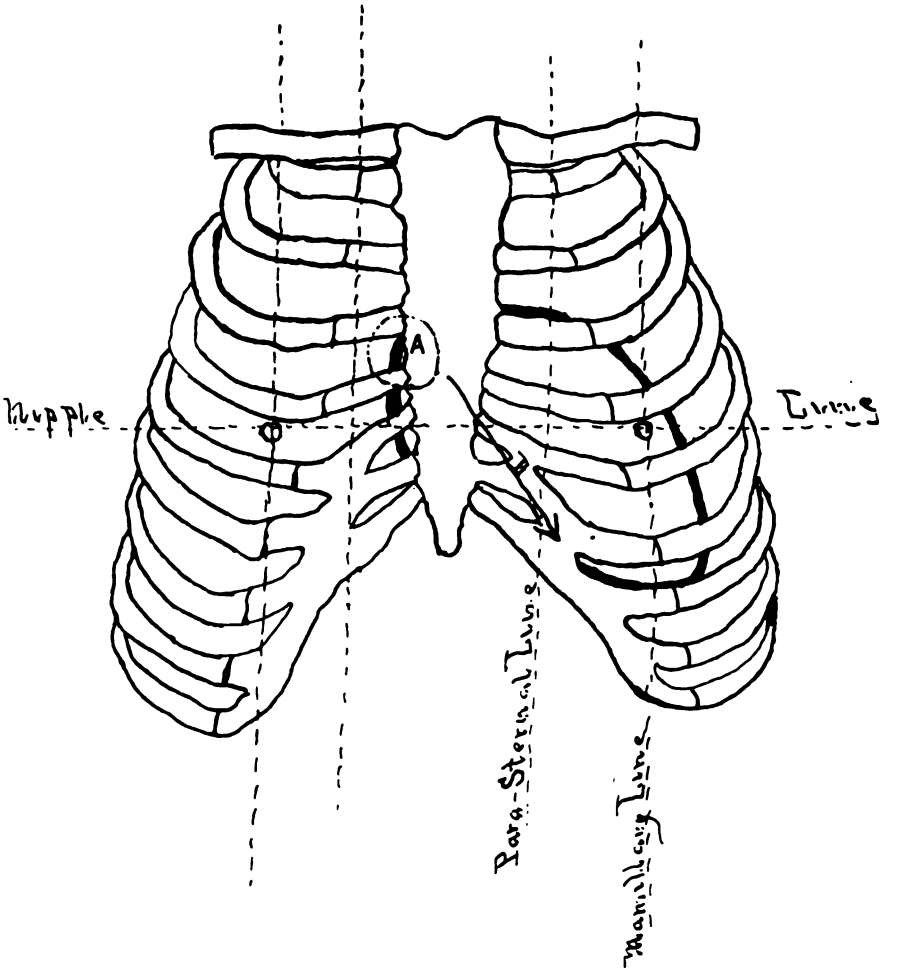


Fig. 57—Enlargement of the heart in aortic regurgitation (Patton). A Area of greatest intensity of murmur, somewhat depressed. B Direction of transmission of murmur.

perfectly distinct here, even when absent over the valve. Broadbent considers this a favorable sign, indicating that the amount of regurgitation is small.

Other Symptoms. Cardiac palpitation, distress and faintness on sudden exertion or rising, are very early features, due to arterial anæmia. Pain and even angina are frequent. Headaches, dizziness and attacks of syncope from disturbed cerebral circulation are not uncommon and likewise may be early manifestations.

Nocturnal dyspnœa and œdema of the ankles and under the eyes indicate failing compensation. The patient sleeps at first with the head elevated, then in a chair, lastly at a table. The sleep is fitful and disturbed by a sense of impending suffocation. General dropsy is not a terminal complication unless mitral disease is associated. Endocarditis, emboli, paralysis and hæmaturia may each play its part.

It is in aortic incompetency that sudden death frequently occurs, due in many cases to sudden over-distention of the left ventricle. In others, the case progresses through the stages of venous congestion and pulmonary engorgement to general dissolution.

AORTIC STENOSIS.

Pure aortic stenosis is the least frequent of the valvular affections of the left side of the heart. In almost every case of stenosis there is some incompetency. The majority of the cases are found in old men, associated with extensive atheromatous changes of the arteries, and the older the subject the greater the likelihood of there being calcareous deposits within the aortic ring. Uncombined aortic stenosis is the least dangerous of the various forms of valvular disease.

The first effect of the stenosis is hypertrophy of the left ventricle. Since there is no over-filling or backward flow in this lesion, simply a demand for increased power of propulsion, the hypertrophy comes about with little or no dilatation. The cavity walls thicken enormously without enlargement of the chamber. As long as compensation is perfect the change is confined to the left ventricle. With failure of compensation dilatation occurs, then its auricle suffers and involves the right heart in the same order as was involved the left. With the left ventricular dilatation comes enlargement of the mitral ring, and relative incompetency of that valve.

Inspection. Even when considerable hypertrophy is present the apex impulse may be invisible, or if visible, may be feeble. More often, however, it is slow, forceful and heaving. It is displaced downward and outward into the sixth or seventh interspace.

In old men considerable emphysema of the lungs is present and

serves to obscure the impact as well as to diminish the area of dullness.

Palpation. The impulse may be impalpable and feeble, or forceful owing to causes given. The chief diagnostic symptom is the presence of a well-marked thrill felt over the base of the heart, and centering over the aortic region. It occurs with systole and may be of great intensity. In some cases it may be felt with diminished intensity over the apex. (Anders.)

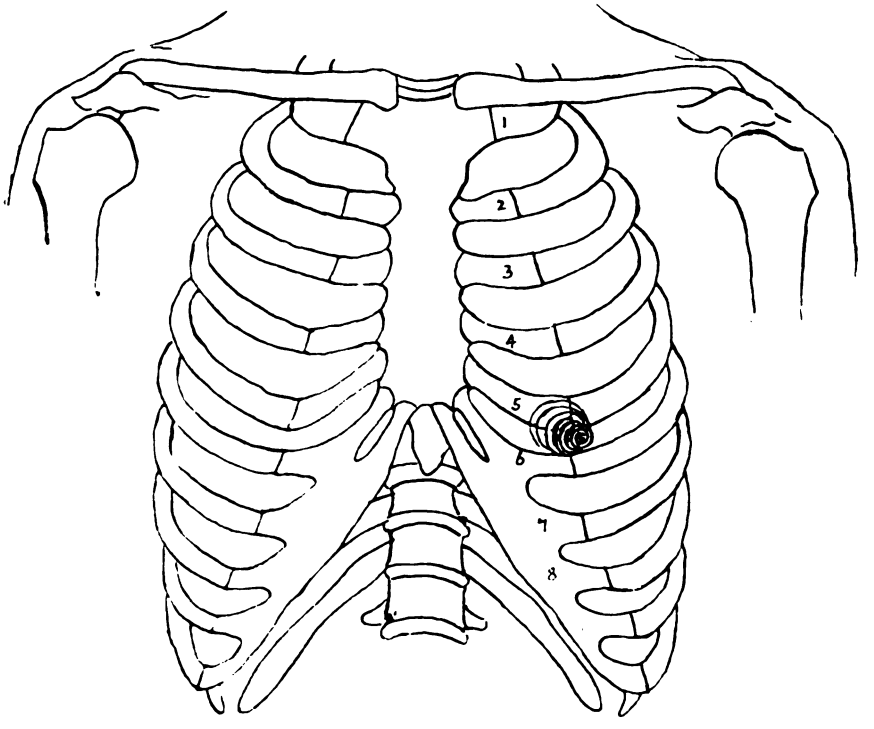


Fig. 58—Mitral presystolic murmur, area and direction.

Percussion. The area of dullness is increased transversely, but never to the extent found in aortic insufficiency. Usually the area as marked out by percussion falls much within the true cardiac boundaries, owing to the masking of the dullness by the emphysema.

Auscultation. A harsh, loud, prolonged murmur is heard, synchronous with each systole. Its point of greatest intensity is either the second right or the second left interspace close to the sternum. The second sound is obscured or more generally absent, since the lesion so

thickens and stiffens the valve leaflets as to prevent their closure. As the ventricle loses power with dilatation, the murmur softens in tone and late in the disease may disappear.

Associated leakage and roughening are manifested by the sometimes present diastolic murmur causing a see-saw sound. Although the conditions necessary for the production of this see-saw murmur are very frequently present, yet it is so often masked by the systolic thrill that it is inaudible. The systolic thrill is propagated into the great vessels. When mitral incompetency supervenes, it is indicated by a murmur, also systolic, sometimes musical, which is heard at the apex. Tricuspid involvement manifests itself in the usual way.

The pulse is the pulsus tardus; small, well-maintained in tension but slower than normal. The accompanying tracing shows its character.

Pulse curve of aortic stenosis—note absence of aortic notch.

Curve of mitral stenosis.

Fig. 59.

Diagnosis. These findings point to aortic stenosis:—

A rough, loud, musical systolic murmur of maximum intensity at the aortic cartilage.

A thrill felt at the base.

Signs of hypertrophy of the left ventricle.

The pulsus tardus.

The mistakes are due to anæmic murmurs which are much softer, more apt to be found in the young, often intermittent, unaccompanied by thrill, and *disappear under treatment.* In anæmias, the second aortic sound is accentuated.

Bright's disease produces cardiac enlargement with which arteriosclerosis is commonly associated, but the urinary symptoms point to the disease, and in the aortic calcification of this condition the second sound is not absent, as it is in stenosis.

Other Symptoms. Cerebral anæmia during the failing stage brings on attacks of syncope or dizziness, often of the most distressing

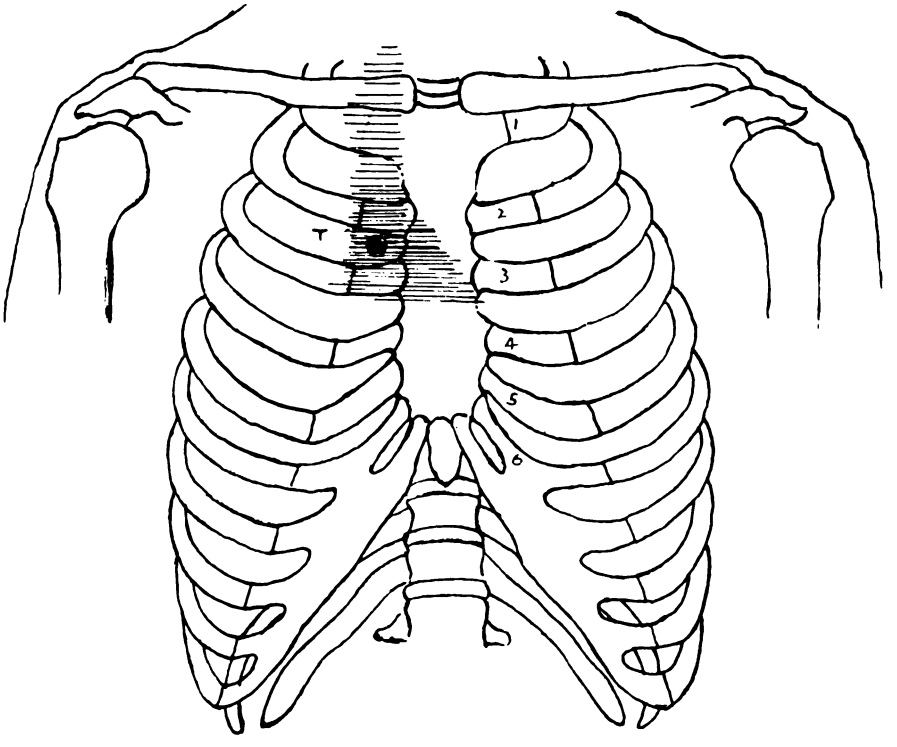


PLATE XV.

Aortic Stenosis. Seat of Greatest Intensity. T, Thrill. This Murmur may be Audible all over the Chest and in the Great Vessels.

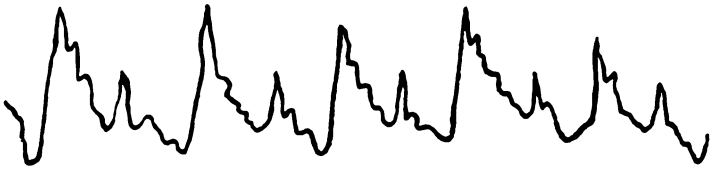
character. In a case under observation the dizziness is *centripetal*, that is the patient always describes an ambulatory circle to the right and falls towards its center.

General œdema and dropsy are rare. Œdema of the feet occurs. The other symptoms are due to associated lesions.

AORTIC STENOSIS AND INSUFFICIENCY.

As stated by Broadbent, there are few if any cases of aortic stenosis without some insufficiency and the double aortic lesion is by many writers ranked next in frequency to mitral incompetency, hence a double aortic lesion is more frequent than a single one. Hypertrophy of the left ventricle reaches the same mammoth proportions as in the single lesion and the vascular phenomena are little modified.

Mitral disease. Compensation fairly well maintained. Inequality of force of successive beats shown—insufficiency slight.



Combined aortic and mitral disease, hypertrophy marked, pulsus alternans.

Fig. 60.

The double aortic lesion is evinced by a double murmur, systolic and diastolic, best heard over the base, on both sides of the sternum, and named, from its striking similarity to the sound of that vessel when afar off, the steam-tug murmur.

The combination of aortic stenosis and mitral insufficiency gives rise to synchronous systolic murmurs, one centering in the apical region, conducted across the axillary space toward the scapular angle, the other best heard at the aortic cartilage, conducted into the vessels of the neck.

TRICUSPID REGURGITATION.

The causes may be grouped under three heads:

I. *Inflammatory.* Acute or chronic endocarditis with resulting

deformity—occasional. Fœtal endocarditis is more prone to attack the right than the left side. According to Rauchfuss endocarditis is more common on the right side of the fœtal heart only because the valves are there most often the seat of developmental errors.

II. Valvular lesions of the left heart producing relative insufficiency by the ensuing obstruction to the pulmonary circulation—more common.

III. Obstruction to the pulmonary circulation occasioned by cirrhosis, fibroid phthisis, emphysema and chronic bronchitis—most frequent.

The ventricle first hypertrophies and then dilates. The blood is projected into the right auricle and thence into the veins of the neck. The strength of the venous pulsation indicates the force of the regurgitation. When the dilatation and insufficiency are great the pulsation is strong. The right external jugular best displays the throb, but the subclavian and axillary veins may also show it. It is best seen when the patient is semi-recumbent. It is greatly intensified by coughing and is an important physical sign.

Inspection. In addition to the signs of overloaded veins and venous pulsation an impulse is seen in the epigastric region, or on one or other side of the ensiform cartilage, which is caused by the hypertrophied ventricle. Holding the breath, coughing or blowing increase its force as well as that of the venous throb. As already explained, hypertrophy of the right ventricle lifts the left ventricle away from the chest wall, so that the apex beat on the left is invisible. The pulsating liver may be visible.

Palpation. The above-mentioned signs are confirmed. Liver pulsation is less often seen than felt.

Percussion. The area of dullness is increased to the right of the sternum. If the disease is secondary the increase is also to the left.

Auscultation. A systolic murmur having its greatest intensity in the lower sternal region is heard. The murmur is soft, blowing and of low pitch, and while less widely transmitted than the corresponding mitral murmur, is often heard in the right axilla. Even when associated with a mitral systolic murmur the softer quality and lower pitch of the tricuspid bruit enables one to separate the two sounds. The co-existence of venous pulsation with this bruit may be taken as *positive* indications of the lesion.

False murmurs seldom attach themselves to the tricuspid valve

and physiologic venous pulsation is not systolic. It corresponds with auricular and not ventricular contraction.

Diagnosis. The diagnosis of tricuspid insufficiency presents no difficulties, and with these three points in mind a positive opinion may be given.

Increased area of dullness to the right of sternum.

Systolic murmur centering at xiphoid.

Venous pulsation in external jugulars.

The tricuspid murmur which develops secondarily to a mitral systolic murmur, owing to its softer quality and lower pitch, may be inaudible. In that case the venous pulsation may be taken to indicate the lesion, especially if hepatic pulsation co-exists. Asystole sometimes develops and portends evil.

Other Symptoms. The symptoms are those of the allied heart lesions or of the co-existent bronchitis, emphysema and pulmonary cirrhosis. General venous engorgement and universal anasarca prevail. The kidney and liver engorgement are followed by a complex symptom train.

TRICUSPID STENOSIS.

The disease seldom exists as an isolated affection. Congenital cases die young. Women are afflicted four times as often as men. It is secondary to lesions of the left heart. The mechanism is exactly like that of mitral stenosis with which lesion it is oftenest associated.

The physical signs are not always well-defined.

Inspection. Cyanosis, especially of the face and lips, is generally present.

Palpation. Even when present the thrill can rarely be felt.

Percussion. Slight change in cardiac area, a little increase to the right of the sternum.

Auscultation. Thrill may be present and is presystolic. A presystolic murmur is often to be made out in the costal angle at the right of the ensiform cartilage, sometimes in the fourth or sixth right interspaces. Either the murmur is often absent or the murmur of the associated mitral stenosis, of which it forms one of the most serious complications, so obscures it that it is indiscernible. General anasarca, extreme and obstinate, is an end symptom.

Diagnosis is difficult owing to the masking of the bruit. It is

seldom made. The presence of the thrill is convincing. Venous repletion and dropsy suggest it.

PULMONARY VALVE DISEASES.

These lesions are rare. Under the caption False Murmurs are mentioned some of the conditions which are mistaken for pulmonary valvular diseases. Care must be taken not to mistake the transmitted mitral murmur mentioned under that disease, which occasionally centers around the root of the pulmonary artery, for disease of this valve.

Pulmonary insufficiency is the rarest of all valvular defects. It is generally congenital.

Inspection. Constant cyanosis, great venous congestion and dyspnœa are marked symptoms. Jugular pulsation is mentioned by Thompson.

Palpation—negative.

Percussion. Hypertrophy of the right heart, of moderate degree.

Auscultation. The lesion should be evidenced by a diastolic murmur of maximum intensity in the second left intercostal space. The corresponding murmur of aortic insufficiency theoreticaly centers on the opposite side of the sternum, but in fact the differentiation by position is impracticable and in any case is extremely difficult. The rarity of the one contrasted with the frequency of the other affords ample grounds for error if not for excuse.

The strongest differential point is that the vascular phenomena accompanying aortic insufficiency, are absent in the pulmonary disease.

Pulmonary stenosis, also congenital, is caused by union of the valve segments. Cases are occasionally encountered in which vegetations are the cause of both insufficiency and stenosis (Snattuck). The congenital cases are associated with compensatory lesions of the foramen ovale or ductus Botalli, or imperfect ventricular septum due to arrest of development.

Inspection: Negative.

Palpation. There may be a thrill in the second left intercostal space.

Percussion. Slight enlargement of right heart.

Auscultation. There should be a systolic murmur with point of greatest intensity to the left of the sternum in the second interspace.

This murmur is not transmitted into the vessels of the neck. The

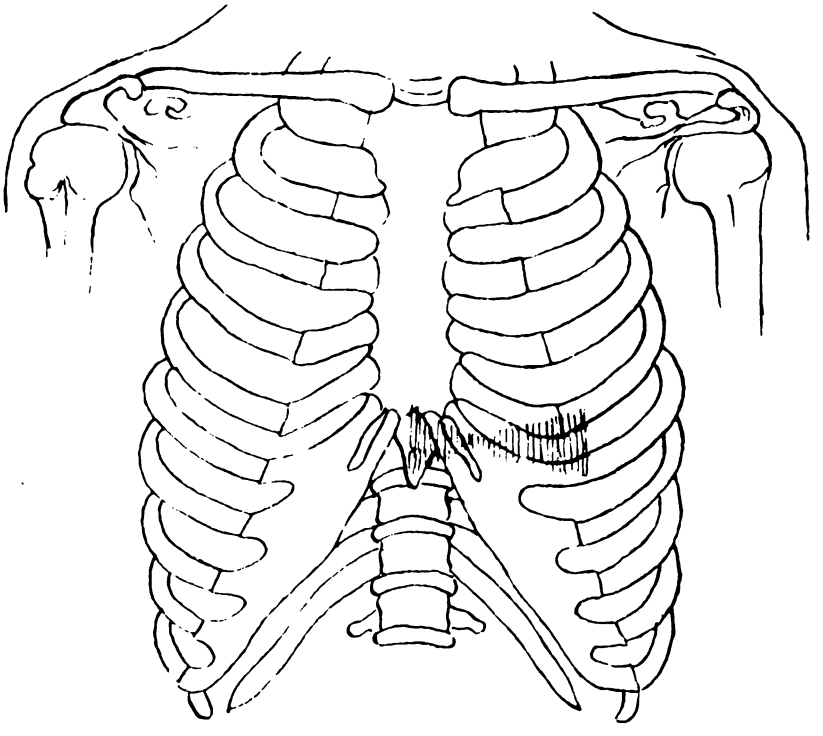


PLATE XVI.

Mitral and Tricuspid Systolic Murmurs. The "Hour-Glass" Murmer.

pulmonary second sound would be absent or replaced by a diastolic murmur, since incompetency is necessarily associated.

The differentiation from the murmur of aortic stenosis presents difficulties. The left-sided hypertrophy is absent in the pulmonary form.

ORDER OF FREQUENCY OF THE SIMPLE AND THE COMBINED CARDIAC LESIONS.

Statistics differ materially as to the order of frequency of both simple and combined lesions. The following order is as nearly correct as available figures permit:

1. Mitral incompetency.
2. Aortic incompetency.
3. Mitral stenosis.
4. Aortic stenosis.
5. Tricuspid stenosis.

The claim of mitral incompetency to supremacy is undisputed. Aortic incompetency and mitral stenosis are practically equal in frequency, and the same may be said of the two forms of tricuspid disease. Of double murmurs, heard at the same orifice, the mitral lesions are more frequent than the double aortic murmurs.

The combined lesions occur in the following order of frequency:

The mitral and aortic segments are most often affected together.

Next in frequency is the combination of mitral and tricuspid lesions; then of aortic, mitral and tricuspid.

In children the most common combination is mitral insufficiency with aortic insufficiency. In adults mitral insufficiency with aortic stenosis and insufficiency is perhaps the oftenest found combination.

The following order is taken from the statistics of F. J. Smith:

1. Aortic incompetency and stenosis: Mitral incompetency.
2. Aortic stenosis and mitral incompetency.
   Aortic incompetency and mitral incompetency.
   Equal.
4. Aortic incompetency and stenosis; mitral stenosis and incompetency.
5. Mitral incompetency and tricuspid incompetency.
6. Aortic incompetency and stenosis; mitral incompetency, tricuspid incompetency.

CONGENITAL CARDIAC DEFECTS.

Two causes are recognized: arrested development and fœtal endocarditis.

Acardia, double heart, dextro-cardia and malposition of the heart are pathologic curiosities. Defective auricular or ventricular septum is more common. When the opening is very small or slit-like little harm seems to result. Patent foramen ovale, imperfect septum and persistence of the ductus arteriosus Botalli each exists alone or combined with stenosis of the pulmonary valve. Patent foramen ovale is sometimes found post-mortem when the defect has been unsuspected.

Peacock states that the most frequent conditions are:

1. Stenosis and atresia of the pulmonary valve, the pulmonary artery and the right conus arteriosus. In 181 congenital cases he found one or the other in 119.
2. Defective auricular septum.
3. Defective ventricular septum.

These may exist alone, but are more frequently associated with pulmonary stenosis, perforate foramen ovale, and patulous ductus Botalli, in the order named.

4. Congenital lesions of the tricuspid orifice, pure or associated.
5. Persistency of the ductus arteriosus.

When existing alone, may be found at an advanced age.

6. Congenital narrowing of the aortic orifice.
7. Congenital narrowing of the mitral orifice.

The number of valve segments is sometimes increased or diminished. Increased pulmonary and deficient aortic leaflets occur oftenest. Two segments may be fused, in which case thickening as a rule coexists. Bicuspid aortic valve has been oftenest noted. The tendency to valvular disease later in life is increased by these defects.

Fœtal endocarditis leads to sclerosis and thickening of the edges and to shortening of the chordæ tendineæ. In some cases the results resemble a perforate diaphragm, especially in stenosis of the pulmonary valve. The valve may be rough or perfectly smooth. Smoothness is taken to indicate imperfect development. Roughness indicates endocarditis. The subjects often die of tuberculosis. Defective septum may be associated. Congenital aortic defects are less frequent than pulmonary lesions.

Symptoms. The most striking symptom of congenital defect of

the heart is cyanosis, which appears soon after birth and is permanent. It may be of slight grade, affecting only the lips and finger tips, ears and toes, and only come on after crying; or it may be of high grade, the entire body exhibiting a bluish or purplish lividity. Clubbing of the fingers and toes is usual and extreme and the nails sharply curve. Cough and dyspnœa, both increased by slight exertion, are generally present. A systolic murmur heard over the auricles and absent at the base indicates the lesion. In congenital pulmonary stenosis the second pulmonary sound is abnormally weak or absent.

DISEASES OF THE MYOCARDIUM.

Atrophy of the heart is the reverse of hypertrophy. Diminution in the size of the heart keeps pace with diminished need. It is congenital or acquired. Wasting diseases and old age are causes of the acquired form. The wasting which accompanies phthisis is a well-known example.

Physical Signs. Weakened impact, usually invisible, small weak pulse, diminished cardiac area. In estimating the latter the influence of emphysema, so often present in old age, must be kept in mind. The sounds are generally weak but may be otherwise normal. In phthisis the second pulmonary sound is accentuated despite the atrophy.

HYPERTROPHY AND DILATATION.

Cardiac enlargement occurs in three forms:

1. Simple hypertrophy, in which the walls are thickened without any change in the size of the cavities.

2. Hypertrophy with dilatation; dilatation with hypertrophy or excentric hypertrophy, in which both dilatation of the cavities and thickening of the walls are present.

3. Simple dilatation, or dilatation of the cavities with thinning of the walls. It is manifest that if the walls are stretched, they must thin.

The weight of the heart has been given. The normal thickness of the left ventricle is from one-third to one-half inch; the right ventricle from one-sixth to one-fourth of an inch; the auricles are about a line in thickness.

The thickness of the left ventricle has been known to reach an inch and a-half, the right, three-fourths of an inch in thickness. The auricles may double their normal thickness in extreme cases.

The weight of the heart in these cases varies from fifteen to twenty-five ounces, except in such extreme cases as follows: Stokes reports a heart of sixty-four ounces; Alonzo Clark, one of fifty-seven ounces; Beverly Robinson, one of fifty-three ounces.

Independently of cardiac valvular lesions there is a small group of hypertrophies and dilatations which deserves notice. They are sometimes designated as idiopathic, as opposed to the hypertrophy caused by valvular diseases. Either the hypertrophy or the dilatation may be primary, but eventually the one condition brings about the other, hence ultimately they co-exist.

Causes. Increased labor and increased intracardial pressure. The part affected depends on the part called upon to bear the strain. Mechanical obstruction to the circulation, if produced suddenly, brings dilatation, if brought about gradually, hypertrophy supervenes. The left ventricle is primarily affected in those conditions in which the impediment is in the general arterial circulation. Such are arteriosclerosis, increased intra-thoracic pressure. A moderate degree of hypertrophy exists during pregnancy, according to Larcher. Immoderate athletic exercise, continued use of alcohol, particularly beer (Strümpell), and all forms of Bright's disease. The right ventricle is primarily affected when the causes are chiefly the outcome of impeded pulmonic circulation, and is affected secondarily in diseases which have produced the same condition in the left heart. We are of the opinion that also associated action and nerve supply is responsible for a considerable amount of concurrent hypertrophy in these cases as well as in valvular diseases. Nerve and muscle fibers are in large part common to both hearts and ability to disassociate their work must needs be an acquired function.

Right hypertrophy is seen as a result of undue muscular exercise in athletes and soldiers (Da Costa).

The lung group embraces emphysema, cirrhosis, phthisis with consolidation, and pleuritic adhesions. Yet left hypertrophy quite as often as right exists in these cases.

Hypertrophy of the heart as a whole is due to associated causes, those which call for increased work of both sides: Exophthalmic goitre, certain diseases of the nervous system, excessive use of cardiac stimulants,—tea, coffee, and alcohol,—pericardial adhesions, as asserted by Quain, independent of valvular diseases. Sometimes pericarditis is the cause of extreme dilatation without hypertrophy.

In cases where dilatation exceeds hypertrophy, some form of myocardial degeneration generally will be found to co-exist.

Sudden death with acute dilatation in infective diseases, and death occurring after slight exertion during convalesence, are probably so caused. The dilatation found in old alcoholics and in cases of widespread arterio-sclerosis is associated with fatty infiltration, fatty degeneration or fibroid overgrowth. Dilatation which supervenes upon hypertrophy in valvular diseases is probably due to the proneness of the hypertrophied muscle to undergo degeneration.

Simple dilatation is most often observed in the auricle and the right ventricle.

Symptoms and Physical Signs. As long as the balance is maintained between the two conditions few symptoms present themselves. There may be some dyspnœa, and vascular throb may be present in the neck. In failing compensation the vascular phenomena described under cardiac lesions supervene, venous repletion and congestion of the various organs. Headache, giddiness and palpitation are frequent.

Inspection. Præcordial bulging, especially in children, cardiac impulse displaced outward and lowered to the sixth or seventh interspace. Dilatation is indicated by the diffused, wavy, uncertain stroke.

Palpation. The impulse may be double, one systolic, one diastolic. Pulse regular, full, tense in hypertrophy; weak, irregular and more frequent in dilatation.

Percussion shows area of increase.

Auscultation. In hypertrophy the first sound is dull, heavy, prolonged and sometimes reduplicated. Laennec called attention to a click, sometimes heard in the young.

In dilatation the quality of the first sound approaches that of the second, the pause shortens and we have a simulation of the fœtal heart sounds, called by the French, embryocardia. Frequently the canter rhythm, or *bruit de galop,* is present. Murmurs are absent unless the condition results in valvular incompetency.

Diagnosis. Four points are insisted upon:

Increased area.

The heaving or diffused impulse.

Absence of murmur.

Character of the sounds.

Acute myositis, abscess of the heart, aneurysm of its walls, fatty

changes, acute myocarditis, give rise to no distinctive signs in themselves, but may bring about results which are recognizable.

Fibroid heart (myocarditis) is followed by a train of symptoms due to the resulting dilatation and venous congestion. It is usually a part of endocarditis. Its physical signs are those of dilatation. A variable and unstable mitral murmur may be present without accentuation of the second pulmonic sound.

ACUTE ENDOCARDITIS.

Definition. Inflammation of the lining membrane of the heart. The valves are usually involved, though the lining membrane of the cavities and the chordæ tendineæ may be affected alone. That the latter condition is not as rare as might be supposed is proved by the statistics of the Royal College of Physicians, in which series of cases the walls were affected 33 times in 209 cases.

It is usual to distinguish two varieties of the disease, a simple and a malignant form, but in reality the distinction is one of degree rather than of anatomical difference.

Simple or benign endocarditis always arises in the course of some other disease. Rheumatism plays the chief rôle, with pneumonia second. Tonsillitis and scarlet fever are liable to the complication. Other infectious diseases less frequently show it, but the possibility of its occurrence as a late complication must not be forgotten. Attacks during the course of phthisis are not rare. The causal relationship of chorea to endocarditis in early life can not be denied, and a large proportion of patients who have been afflicted with the former carry the lesions of the latter malady.

The malignant type may exist as an independent disease, but secondary forms are more usual. In infective diseases, pneumonia and kidney diseases the malignant variety is prone to occur.

While in rheumatism, as in pneumonia, the benign type is oftenest met with, yet the malignant form is not rare in either disease.

Septic infective diseases furnish a proportion of the cases.

Symptoms. Simple endocarditis gives rise to no distinctive symptoms or physical signs. In the course of inflammatory diseases increased rapidity and irregularity of the heart's action and increased fever without assignable cause are suspicious.

A murmur at the apex may develop but its presence does not indicate the disease, *nor does its absence mean exemption.* The stu-

dent must free his mind of the widespread fallacy that heart affections must needs be attested by murmurs.

The malignant form is manifested by rigors, sweats, irregular or intermittent fever, delirium and signs of septic infection, which are in no way distinctive of heart involvement. Another type is the typhoid, which is self-explanatory.

In persons known to have chronic valvular lesions, symptoms similar to the above should excite grave apprehensions of a recurrent endocarditis. Embolic symptoms often occur and aid the diagnosis. The disease is perhaps most often mistaken for typhoid fever. Widal's reaction excludes the latter.

SECTION XI.

THORACIC ANEURYSM.

An aneurysm is a more or less circumscribed dilation of an artery. The dilation may include all the coats of the artery, or it may be that one or two coats having yielded or ruptured, the remaining part dilating thus forms a blood sac which communicates with the parent vessel.

A convenient division of these lesions is into internal, or medical, and external, or surgical, aneurysms, which classification is self-explanatory.

Aneurysm of the thoracic portion of the aorta is by far the most frequently met with among aneurysms, since aneurysms of the aorta follow the same law as does sclerosis with regard to situation, viz., the nearer the heart the greater the pressure, hence the greater the liability to aneurysm. For this reason they are oftenest found in the ascending portion of the arch, which is the subject of attack five times as frequently as the abdominal aorta; then in the transverse and descending portions, where they occur three times as often as in the abdominal vessel. For the same reason they are found on the convexity of the arch, rarely on the concave side.

Anatomy. The aorta lies in the middle mediastinum. It arises from the base of the left ventricle, behind the left margin of the sternum, opposite the lower border of the third left costal cartilage. This point corresponds to the body of the fifth dorsal vertebra. It passes upwards, forwards and to the right, between the superior vena cava and the pulmonary artery, as far as the right border of the sternum, beyond which it does not project, and as high as the second costal cartilage. Its breadth is 28 m. m. At the point given it arches upwards, backwards and to the left, winding around the trachea to reach its left side, whence it passes to the left side of the lower border of the fourth dorsal vertebra, where it becomes the descending aorta, which ends at the opening of the diaphragm. The trachea lies first behind the aorta, then to its right side, within the curve of its arch.

Bifurcating at the lower border, the left bronchus passes beneath the arch. The left phrenic and pneumogastric nerves pass in front of the arch; the left recurrent laryngeal winds under it; the right pneumogastric passes behind it. The œsophagus lies behind the arch at first, between the trachea and the border of the vertebræ, then to the right of the descending aorta.

Inspection in the early stages reveals nothing. Sooner or later, however, a fullness is apparent over the anterior portion of the chest above described, varying with the seat and size of the tumor. It may be merely a fullness, or it may be a prominent elevation of one or more ribs, the inner end of the clavicle or of the sternum. The bulging most frequently occurs to the right of the sternum in the second or third interspaces, or even as high up as the end of the clavicle if the tumor be of the ascending limb, or lies behind the manubrium, when the transverse arch is affected. Tumors of the descending arch may cause bulging to the left of the sternum, but are less likely to do so until they have attained a very considerable size, owing to the greater depth of the descending portion of the structure.

Palpation reveals an area more resistant than the surrounding region, and a distinct pulsation may be communicated to the superimposed finger-tips which seems to come from beneath. In advanced cases this impact gives the sensation of dilation or expansion of the tumor mass; at other times a sensation which is best described by the word "heaving." It is almost synchronous with cardiac systole, but is differentiated from the heart by its character and location. It may be feeble, or may seem stronger than the heart-beat itself. Its location is directly behind the sternum opposite the second interspace, or is felt in the second interspace, either to the right or to the left of the sternum, being a little lower on the right side than on the left. In case calcification of the sac has taken place this expansile impact is wanting, as the elasticity of the sac is thereby destroyed. Where a laminated clot partially fills the sac the same observation applies in varying degrees; but in both cases impact often remains.

Percussion reveals an area of dullness corresponding to the enlargement. Its situation is that of the aorta, and lies higher than the base of the heart, from which it is sometimes separated by a narrow isthmus.

Auscultation. Sometimes nothing is heard over the tumor except the transmitted heart sounds. Both sounds may be heard, or only the

first sound, which is then generally accentuated and prolonged. Careful comparison should be made with the sounds heard over the apex beat as to the time and duration of the aneurysmal sounds. Two other sounds are intimately associated with aneurysm, and while their presence means much their absence does not indicate that the tumor is not aneurysmal. These sounds are the thrill, which is more apt to be present when the vessel is the seat of extensive atheroma; and the bruit, which is the name given to the whirl of blood within the dilation or sac. This sound, which is the one most commonly heard over an aneurysm, is synchronous with systole, generally single, and is of short duration.

The most positive single sign, therefore, of aneurysm, is an impact or pulsation within the above-described area, which is independent of the heart and which is accompanied by its own sounds. These sounds are superficial in character, unlike those produced by a new-growth pressing downward from the surface upon the aorta, in which case also a deep-seated murmur may be heard over the course of the aorta.

Fluoroscopic exmanination and radiographs often make certain the diagnosis of aneurysm when the physical signs fail to reveal its presence, as noted in Section XVII. This is especially the case when clot, partly obliterating the sac, modifies, or causes to disappear most of the auscultation signs; whereas the solidification renders the tumor more apparent in the radiograph.

Pressure Symptoms. There is but little unoccupied space within the chest, and any enlargement, whether it be of existing organs or a new-growth, is sure to trespass upon some of its neighbors and give rise to pressure symptoms. The diagnostic value of this symptom group will be appreciated when we consider Bramwell's ornate classification of aneurysms into three groups:—aneurysms which are latent and give no physical signs; aneurysms which present signs of intrathoracic pressure, but not sufficiently definite to determine the nature of the lesion; aneurysms which produce distinct tumors, physical signs and pressure-symptoms.

From their nature and situation, aneurysms of the arch are most prone to press upon the trachea, giving rise to dyspnœa, cough and shortness of breath. The cough has a peculiar ringing quality which may be mistaken for chronic affection of the larynx. It may be dry, or may be accompanied by profuse watery or muco-purulent expectoration, by râles and other signs of bronchitis. The voice may be

stridulous, hoarse and croaking, and the sentence often ends in a peculiar gasp. Both inspiration and expiration are equally affected. If the œsophagus be pressed upon, we get dysphagia. Inequalities of the two radial pulses are not infrequent, due to pressure upon the subclavian. Sometimes inequalities of pupils are noted. These signs may be present in case of mediastinal new-growths, but in such case the pulsation is absent. Furthermore, aneurysm is by far the most common intra-thoracic tumor. (Symptoms caused by mediastinal growths are considered in a separate paragraph.)

Pain is a prominent and frequently an early symptom. At first the pain may be fugitive but gradually becomes more and more constant and varies in intensity and character with the structures pressed upon. Violent, recurrent pains within the thoracic cavity, especially if shooting up into the neck and arms, or pain of a boring character, should excite the suspicion of aneurysm. Men are much more frequently attacked than women, and the selective age is between 35 and 45 years.

Aneurysms of the Ascending Portion of the Arch. Aneurysms of this portion of the arch may push the heart downwards and to the left. They involve most frequently the right recurrent laryngeal nerve, the vena cava superior, sometimes the inferior cava, or the right subclavian vein. The result of such involvement is widespread œdema. The innominate artery usually escapes.

Aneurysms of the Transverse Arch. These tumors occupy the middle line or project to the right much more often than to the left. They involve the trachea, left laryngeal nerve and the innominate artery. The radial and carotid pulse may be absent. They may attain enormous size, projecting into the sternal notch. The pressure symptoms vary but correspond nearly to those given. The pupils may be at first dilated, but later contracted or unequal.

Aneurysms of the Descending Portion of the Arch. The sac projects backwards and erodes the bodies of the thoracic vertebræ, or the tumor appears under the left scapula. It gives rise to an area of percussion-dullness in the left spinal gutter, from the third to the sixth dorsal vertebræ. Dysphagia is common.

Aneurysms of the Descending Thoracic Aorta. These occur generally just above the diaphragm. Such a tumor is more difficult of detection than aneurysm of the arch, because the majority of the

pressure symptoms are absent and, by reason of its deeper seat, pulsation can not be detected nearly so early.

The pain is usually of a gnawing character, due to absorption of the bodies of the vertebræ. This is one of the most characteristic symptoms. So situated, the growth may simulate lumbago, intercostal neuralgia or back-ache; the latter, especially in women, is an ailment so common as ordinarily to excite little notice.

Aneurysm of the Innominate Artery. An aneurysm of the transverse portion of the arch may involve the innominate and left common carotid, but the innominate is sometimes affected alone. Here the tumor is situated *above* the second interspace and behind the sterno-clavicular articulation. Most of the pressure signs described are absent. Compression of the subclavian and right common carotid, made with the fingers on the affected side diminishes the pulsation in the tumor.

SPECIAL DIAGNOSTIC SIGNS OF ANEURYSM.

The Tracheal Tug. This sign was pointed out by Surgeon Major Oliver, U. S. A., is frequently present and is a most valuable sign. Place the patient with the head bent slightly forward to relax the tissues of the neck. Draw the cricoid cartilage upwards with the thumb and finger sufficiently to gently stretch the trachea. At each heart-throb a distinct downward movement of the cricoid (tug) will be felt and often seen, synchronous with the systole. The tugging is strongest when the growth presses against the left bronchus. The sign is especially apt to be obvious when the aneurysm affects the transverse portion of the arch, particularly its posterior part, and is said to be absent in aneurysm of the innominate, thus serving to differentiate the two conditions. The anatomic relations given make clear the reasons of its production. It may be the sole sign of the malady, and should therefore always be searched for. It is sometimes present comparatively early in the disease.

H. L. Smith directs attention to a sign which he has found to be present in several cases. "This sign is elicited by combined palpation and percussion as follows: The cricoid cartilage is grasped as is done for tracheal tugging, while an assistant percusses the chest. When normal parts are percussed the palpating hand feels a distant, feeble jar (proximal ends of the clavicles excepted), but so soon as the aneurysmal area is reached, a shock which is both direct and resilient

in nature is felt, somewhat suggestive, as I take it, of the sensation experienced by one when a rubber bag filled with water is simultaneously palpated and percussed."

In the one opportunity afforded me since seeing the above, I have been able clearly to elicit the sign.

Special Symptoms. Pain is characteristic of the disease. Generally constant, it is however subject to severe exacerbations, due to pressure exerted directly on nerves or to stretching of the nerve filaments of the sac. Shooting and darting pains over the involved area, or over a considerable portion of the chest, the neck, the occiput and down the arms, especially the left, to the finger-tips, denote pressure on contiguous structures; while unremitting dull, boring pain denotes that erosion of bone is taking place. This applies especially to tumors of the descending limb, in which the vertebræ are transgressed upon. Aneurysms involving the transverse portion of the arch produce the severest pressure symptoms, because of the narrowness of the antero-posterior diameter of the chest and the important structures in intimate relation with this division of the arch, which are readily compressed as soon as the growth attains any considerable size. Growths of the descending aorta may cause pain in the intercostal spaces which is liable to be mistaken for neuralgia. Anæsthesia of the skin frequently accompanies this pain.

Dyspnœa is due to pressure upon nerve trunks, vessels, lung, bronchi or trachea, and is not necessarily in proportion to the size of the tumor. It is least when the pressure is directly exerted upon lung substance, when physical signs of consolidation and collapse of pulmonary tissue may co-exist with only slight dyspnœa. On the other hand, pressure upon the trachea or on one bronchus will occasion severe dyspnœa, amounting to orthopnœa. Such pressure is indicated by noisy, stridulous breathing, loud clanging cough and expectoration. Various râles, from retained secretions; imperfect expansion of the affected side; diminished tactile fremitus; feeble or absent breath sounds, with slight fever are noted on examination, and may suggest phthisis, as pointed out by Hanot.

Sudden and severe paroxysms of dyspnœa, due to increase of pressure when the sac distends; asthmatic seizures resulting from implication of the vagus, particularly the recurrent laryngeal branch, are common phenomena and may suggest angina pectoris. (Pepper holds that true angina may be associated.)

Laryngeal symptoms are common, such as spasm of the larynx, dyspnœa and stridor, or paralysis of one or both vocal cords, with husky or whispering voice, or aphonia. Pressure on the left recurrent laryngeal nerve causes most of these alterations, viz., imperfect vocalization, aphonia, hoarseness and varying degrees of paralysis of the vocal cords. Pressure on the right side of the trachea does not affect this nerve, and therefore aphonia and dysphagia are absent although the voice may be strident (Tufnell).

Cough. The cough is apt to come on in paroxysms, and is loud, brassy, stridulous or ringing. In an impressive case recently seen the patient could only phonate with *inspiratory* gasps. During the attacks the cough was stridulous but ringing, dyspnœa intense, yet with intervals of relief. The case was verified by autopsy.

Dysphagia is a rarer symptom, but occurs especially in cases of aneurysm of the descending division, exerting pressure on the œsophagus.

Alterations of the pupils are frequently concomitant; dilatation occurring when the cervical sympathetic ganglia are irritated by slight pressure, contraction intervening when the pressure causes paralysis. Hence dilatation is an early symptom; contraction a late one. The phenomenon is limited to the affected side. When exophthalmos occurs it is usually bi-lateral. Unilateral sweating, local areas of flushing, of anæsthesia, paleness, or coldness of the skin are occasionally encountered.

Congestion, cyanosis and particularly œdema result from pressure on the superior vena cava and are frequent. It may affect the head and neck, or be limited to one pectoral or one arm. Such œdematous or spongy masses occurring at the base of the neck, above the clavicles, have been termed "the collar of flesh", and are unmistakable.

Pulse. A very old, yet very valuable, sign of aneurysm is alteration in the pulse, causing delay in the pulse wave or even total obliteration. The sign is valuable owing to the ease and certainty with which it may be tested. Thus, in health the two radial pulses are synchronous, but in aneurysm affecting the innominate artery the right pulse may lag, while one affecting the left common carotid and subclavian will cause the left pulse to be retarded. The retardation may be explained by the temporary interruption of maintained pressure in the blood current, on reaching the empty sac, which acts as a miniature reservoir, and the resumption of pressure as soon as the sac is dis-

tended. Narrowing of the exit from the sac would also cause delay by retarding the blood exit.

The accentuation of the aortic second sound so often noticed in cases of thoracic aneurysm is due to the augmented back-flow of blood against the semilunar valves. The increment to the wave being derived from the aneurysmal reservoir.

Differential Diagnosis. New-growths situated in the mediastinum, enlargements of the heart, effusions into the left pleura causing upward displacements of the heart, may be mistaken for aneurysm. In aneurysm the tumor is *above* the base of the heart and the area of dullness caused by its presence is often separated from the heart by intervening lung or joined to it by a narrow band. Its auscultation sounds are separate from those of the heart and possess different qualities. Effusions, while lifting the heart upward, are not accompanied by bruit or thrill; the heart sounds are feeble and distant, the apex displaced. New-growths give rise to pressure symptoms but other signs are absent.

SECTION XII.

EXAMINATION OF THE BLOOD.

Recent advances made in our knowledge of the causes of disease have been due not a little to the study of the blood. The blood changes which accompany disease are of such momentous import, and their recognition contributes so much of inestimable value to the field of diagnosis, as well as to prognosis and cure, that it is the duty of every physician to be able to recognize these departures from the normal, and to interpret their significance.

The present chapter will consider only the more commonly occurring changes and those which fall within the province of the general practitioner, omitting such investigations as require unusual and costly apparatus or the privileges of a well-equipped laboratory.

Normal Blood Elements. By proper manipulation the blood may be readily separated into a clear fluid, the plasma, and the solid or corpuscular constituents. These are the red corpuscles, or erythrocytes, the colorless corpuscles, or leucocytes, and the colorless discs, or blood-plaques of Bizzozero.

Place a drop of blood on a glass slide, cover and examine with the microscope. The red corpuscles form rouleaux, and their color is pale greenish-yellow. Their average diameter 7.5 μ. The rouleau tendency diminishes when their number is below normal. Red cells, larger than normal (diam. 9.5 to 12.) are called macrocytes or megalocytes, those smaller than normal are called microcytes (diam. 3.5 to 6) while those of irregular shape are called poikilocytes. These may manifest motility. The leucocytes are seen here and there in the spaces between the red rows. Staining shows them to be nucleated and generally granular, irregular in shape, larger than the red cells and capable of locomotion. Their varieties will be considered later.

In addition to the above elements the student should search carefully for abnormal constituents, both within and without the cells, such as pigment granules, malarial organisms, filaria, spirochætes and other parasites.

A
D
A
C
C
B
MAST CELLS
D
EOSINOPHILIC
MYELOCYTES
E
E
POLYMORPHONUCLEAR
LEUCOCYTES
NEUTROPHILIC MYELOCYTES
A
B
POLYNUCLEAR
EOSINOPHILIC LEUCOCYTES.

PLATE XVII.

A material increase of any particular cell-element is spoken of as —'osis, hence the term leucocytosis, microcytosis, etc. Microcythæmia, megalocythæmia, etc., have the same significance.

To give a detailed description of the various cells found in the blood in disease were impossible within the limits of this brief treatise, and would serve a less useful purpose than the accompanying classification with attached nomenclature.

The Red Corpuscles. The red cells are circular, bi-concave discs of greenish-yellow color and non-nucleated. The average diameter is 7.5 μ. The center being thinner than the rim is therefore lighter colored. Abnormal pallor can be detected by practice. There is less variation in the size and shape of the red than of the white corpuscles. Moving cells are seen to change their shape, like elastic bags. Macrocytes are probably regenerative cells; microcytes, degenerative.

The name poikilocytes was given by Quincke to irregular forms sometimes seen elongated, balloon-shaped, rod-like, which occur in severe anæmias and in chlorosis. Crenation, which occurs upon the slide, is the ameboid motion with change of shape, and is *outside* poikilocytosis. The absence of the rouleau tendency is sometimes noticeable, and is seen in pneumonia, hepatic diseases and nephritis. Polycythæmia is an actual increase in the number of red blood corpuscles. It is *relative* when due to a diminution of the plasma, as in diarrhœas and ascites. The opposite condition is known as oligocythæmia and occurs in the anæmias, septicæmia and all wasting diseases.

Normal red cells show little affinity for dyes, differing in this respect from the white cells. In disease, red cells which accept stains are met with, also cells not normally present in health but seen in disease, as the microcytes and the megalocytes which stain readily. Staining with methylene blue shows the granular nature of diseased and degenerated red cells. Cells containing such granules occur in pernicious anæmia, malaria, the leukæmias, and lead poisoning. In the last they are very constant, and, in the absence of the other conditions, saturnism should be suspected.

Nucleated red cells are found in the fœtus and immediately after birth, and at all times in the bone-marrow. Three varieties are described:

(a) Normoblasts.
(b) Megaloblasts and
(c) Microblasts.

The first is a developmental form of the normocyte. In other words it is an immature red blood corpuscle. Megaloblasts, cells of 10 μ or over, occur only in disease. They are not found in healthy marrow. They are seen in anæmias due to intestinal parasites, in grave anæmias, especially when pernicious. Ehrlich considers their occurrence as prognosticating death, except in parasitic anæmia. Microblasts are rarer than the above, they are of small size, imperfect form and their import equally serious. Both forms seem to indicate a return to the fœtal type of blood.

The following terms are used to describe the behavior of all cells towards the various dyes employed:

Basophilic, having an affinity for basic dyes.

Achromatophilic, no affinity for dyes.

Oxyphilic, having an affinity for acid dyes.

Polychromatophilic, an affinity for both basic and acid dyes.

Neutrophilic, having an affinity for neutral dyes.

Eosinophilic, having an affinity for eosin stain.

Chromotrophic elements are stained a different color from that of the dye, as when violet dyes stain red.

The White Corpuscles, or leucocytes, are colorless cells. They are generally larger than the red bodies, more irregular in shape, are nucleated and much fewer in number than the red cells. From their composition they are divided into granular and non-granular varieties, or may be classified as mono-nuclear and poly-nuclear forms. The larger varieties, especially the coarse granular cells, are actively ameboid, the small mono-nuclear variety is devoid of ameboid movement.

Classified in accordance with their origin, two groups may be distinguished:

I. Those from the bone-marrow, the myelogenous group, and

II. Those from the adenoid tissue, the lymphogenous group. This second group comprises the lymphocytes of all sizes.

The first group includes the following:

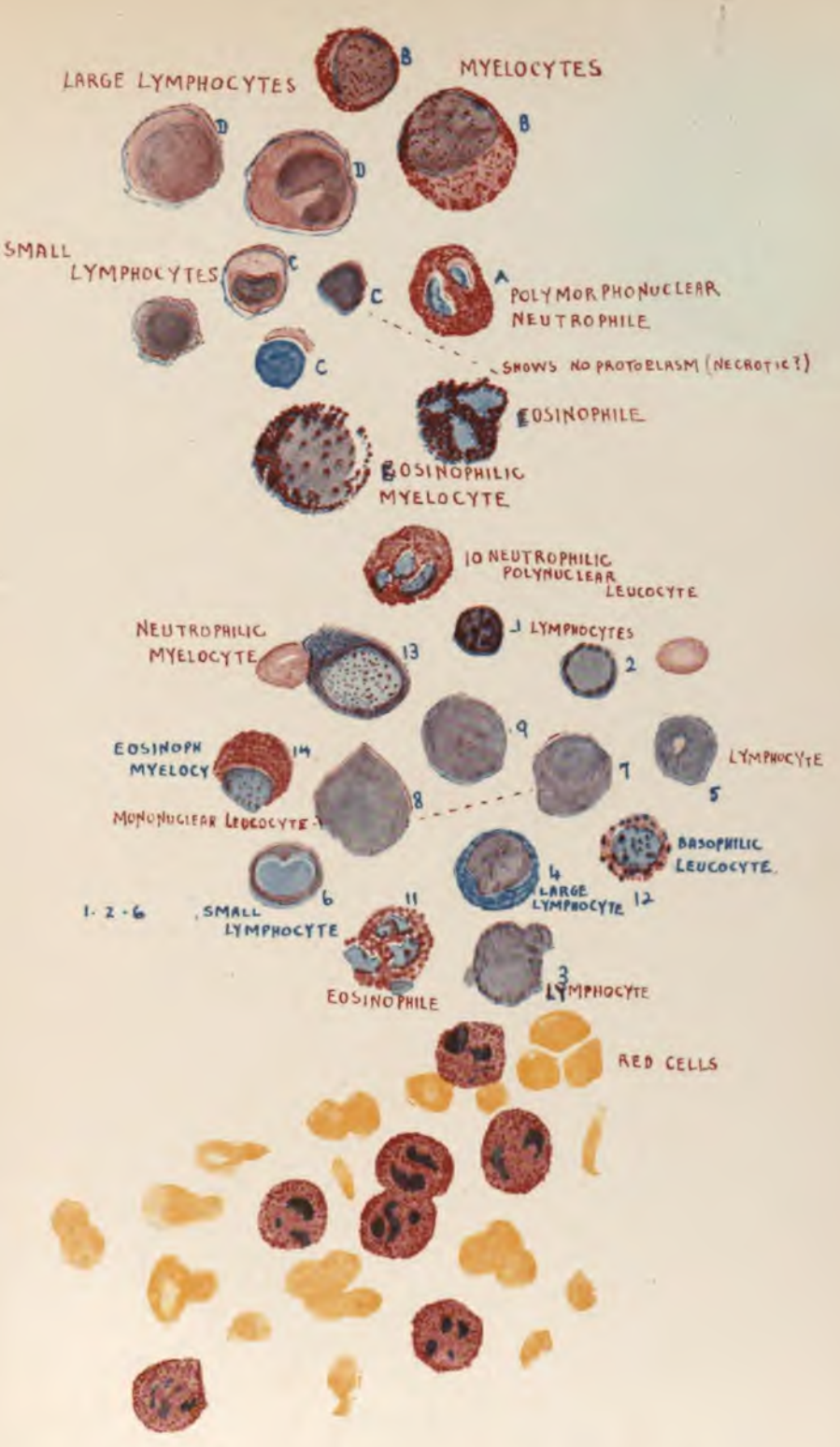
(a) Polymorphonuclear neutrophiles.

(b) Eosinophiles,

(c) Mast cells,

(d) Large mononuclear cells of Ehrlich.

The forms of group I are not transitional.



RED CELLS AND POLYMORPHONUCLEAR NEUTROPHILES

PLATE XVIII.

VARIETIES OF LEUCOCYTES.

*LEUCOCYTES.

Classification according to structure and behavior towards dyes.

A. Non-Granular Varieties. — Mononuclear. Little or no ameboid movement; many contain one or two peripheral granules.

1. Small lymphocytes derived from adenoid tissue. Vary in size from smaller than a red blood corpuscle to considerably larger. Mononuclear, both nucleus and protoplasm basophilic. Outline smooth or irregular. In adults, 20 to 30 per cent. of total number of leucocytes; in infants, 40 to 60 per cent.
2. Large lymphocytes, large mononuclear leucocytes, derived from bone marrow and spleen. Two to three times the size of red blood corpuscle. Large, single nucleus; both nucleus and protoplasm basophilic. Outline irregular, oval. Surrounded by broad zone of protoplasm. Form from 4 to 8 per cent. of total number.

B. Granular Varieties.

I. Polynuclear, polymorphonuclear. Phagocytes, active, ameboid motion. Neutrophilic, basophilic, oxyphilic.

3. Polynuclear nutrophilic leucocytes. Polymorphonuclear neutrophilic leucocytes or phagocytes. About same size as No. 2. Nucleus elongated, twisted, broken. Granules embedded in protoplasm stain with neutral dyes (neutrophilic). Protoplasm proper stains with acid dyes. Most common of all leucocytes. 62 to 70 per cent. of total leucocytes. Derived from neutrophilic myelocyte (Ehrlich).
4. Polynuclear oxyphilic leucocytes, same as No. 3, except embedded protoplasm stains with acid dyes, or eosin, hence also called polynuclear *eosinophilic* leucocytes. 1 to 4 per cent. of total or about 50 to 200 per c. c. of blood.
5. Polynuclear basophilic leucocytes, mast cells, size about same as Nos. 3 and 4. In disease size may reach 22 micro-millimeters. Embedded grains of protoplasm, different sizes, stain only with basic dyes. Nucleus polymorphous. Rare. Never exceed 0.5 per cent.

II. Mononuclear. Granular cells.

6. Myelocytes. Mononuclear neutrophiles, granular cells of the bone marrow, "*Intruders*," occasional occurrence, border cell between physiologic and pathologic conditions. (Starvations and various intoxications.) Are derived from large lymphocytes (2) and develop into the polynuclear leucocytes (3) are never normally found in the circulation. According to action with dyes, are neutrophilic, eosinophilic or basophilic, all with single, large nucleus, surrounded with protoplasm. Diameters 10-20 micro-millimeters.

The protoplasm of normal leucocytes is stained uniformly bright-yellow by iodine. In diseases in which pus is a product of the disease, or in which the infection or intoxication is due to some form of bacterial invasion, as in typhoid fever and pneumonia, the granular protoplasm is stained brown by the reagent. This was announced a few years ago by Kaminer. Later, Wolff announced that the reaction was due to the presence of glycogen and occurred irrespective of disease. Hirschberg has confirmed Wolff's work and the staining can no longer be regarded as significant. The polymorphonuclear neutrophiles best show the reaction, while the eosinophiles are exempt. It was previously taught that the reaction served to demonstrate sepsis and also to distinguish between a serous and a purulent effusion.

The number of leucocytes per cubic millimetre of blood varies much both in health and disease. The average may be stated to be 5,000 to 6,000, but may fall anywhere between 3,000 and 10,000 within strictly physiologic limits. An increase above normal is called

hyperleucocytosis, or simply leucocytosis; a diminution below normal is hypoleucocytosis. The increase or diminution may affect all the varieties above mentioned but more generally it will be found that a particular variety suffers much out of proportion to the remaining cells. The most common form of leucocytosis is an increase of the polymorphonuclear neutrophilic cells (3). In the new-born these exist physiologically to as high as 20,000, which number decreases to 10,000 by the end of the first year. The eosinophiles may reach 2,200 (Taylor). Pregnancy and labor, severe exercise, bathing, and even digestion increase the number of leucocytes above normal. They vary from day to day, and from day to night. Such variations are physiologic and temporary. The number is below normal in the ill-nourished, the sickly and in those who have fasted. If a deficient amount of food has been eaten, as is often asserted by patients, the count will verify the statement. Such conditions of ill-nourishment and unhygienic surroundings must be given proper weight before concluding that a count of, say, 3,000 is pathologic.

Cabot gives the following classes of pathologic leucocytosis:

Post hæmorrhagic leucocytosis.

Inflammatory leucocytosis.

Toxic leucocytosis.

Leucocytosis in malignant disease.

Leucocytosis due to experimental and therapeutic influences.

Among inflammatory conditions in the above classification are included infectious diseases.

In septic conditions leucocytosis increases with the severity of the attack, at least, so long as resistance is normal. Where the resistance is greatly diminished, leucocytosis diminishes therewith, and may fall to a low point. In pneumonia, its absence forebodes a fatal termination.

In many of these conditions, while the neutrophiles are increased, the eosinophiles are correspondingly diminished.

Leucocytosis is absent in the following diseases: Typhoid fever, malaria, grip, measles, rötheln, mumps, cystitis, all forms of tuberculosis and tubercular processes except the meningeal form.

Bloodgood* states that in appendicitis the leucocytosis is a pretty safe index of the severity and extent of the disease. A count of 15,000 falls rapidly to 10,000 with the amelioration of the disease. A count

*American Medicine, 1901, page 306-7.

of 20,000 observed within forty-eight hours of the beginning of the attack demands operation. In gangrenous cases the count reached 25,000 to 30,000. Very high leucocytosis observed within the first forty-eight hours points to peritonitis.

In intestinal obstruction the increase is of especial value, rising rapidly to 20,000 within twenty-four hours, and still higher in gangrene or associated peritonitis. Later in the disease a low count with persistence of the symptoms is unfavorable.

Fevers with leucocytosis are not typhoid, since it does not occur in uncomplicated cases. Its occurrence, therefore, in undoubted cases points to complications. It is stated that in articular rheumatism complicated with endocarditis, the count does not rise much if any above that for the uncomplicated joint affection, viz: 10,000 to 12,000,

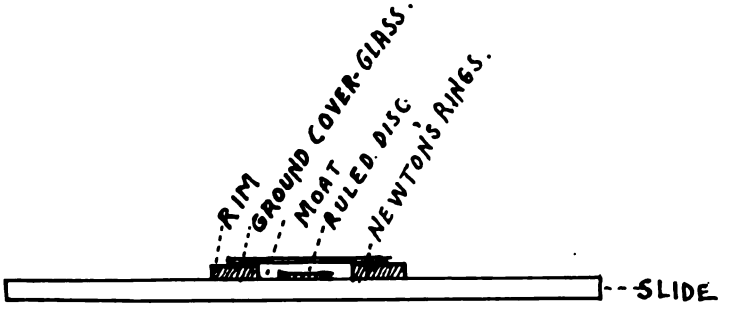


Fig. 61—The Thoma-Zeiss Counting Chamber.

but that when complicated with pericarditis the average rises to 20,000 or over, and that such rise foretells the complication.

Tuberculosis is only attended by leucocytosis when secondary pus infection occurs.

The eosinophiles and the mast cells are increased in myelogenous leukæmia, while in the lymphatic forms of the disease the lymphocytes are increased, numbering 40,000 to 200,000. Increase of eosinophiles is found in bronchitic asthma, in scarlet fever, in hydatid disease of the liver, as noted in one of my cases, in intestinal parasites, gonorrhœa and in inflammatory skin disease.

Blood Counting. The most suitable apparatus for enumerating the blood corpuscles is the cytometer of Thoma-Zeiss, manufactured by Zeiss. It consists of a diluting pipette for the red corpuscles, another for the white, a specially ground cover-glass and a counting

chamber in the bottom of which is placed a disc ruled into squares whose sides measure 1-20 mm. and whose areas are therefore 1-400 of a square millimetre. The depth of the chamber when the cover-glass is in position is 1-10 mm. When the chamber is filled the contents overlying each square will be a column 1-400 mm. base by 1-10 mm. in height or 1-4000 cu. mm.

Hence the number of corpuscles overlying any one square × by 4,000 would equal the number of corpuscles in a cubic millimetre of blood, if the blood examined were undiluted. Since it is impossible to make the count without diluting the blood to 100 or 200 times its own volume, the above product must be multiplied by the dilution multiple. Furthermore, since great variations occur in the number of

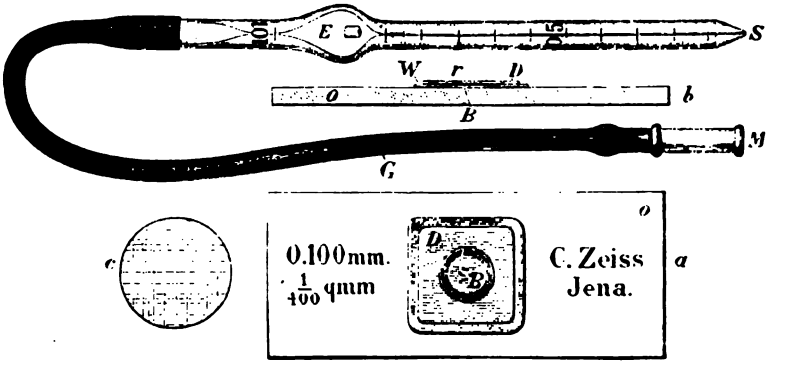


Fig. 62—Thoma-Zeiss Hæmacytometer Pipette and Chamber.

corpuscles found in the various squares, in practice it is actually necessary to count the corpuscles overlying a large number of squares, divide by the number of squares counted in order to obtain an average, then multiply by the other factors. By reason of the ruling presently to be described, it is found convenient to count a field 16×16 or 256 squares. Suppose the number therein totals 1,792, giving an average of 7 for each square, the dilution being 200. Then 7×4000×200= 5,600,000 cells per cu. mm. of blood. Counting chambers with the rulings of Turck enable the enumeration of both red and white cells to be made in the same specimen. In this apparatus the small 1-20 mm. squares are fenced off into blocks of 4×4 or 16 of the smaller squares, separated by interspaces or "alleys," the width of the small squares. For enumerating the red discs, the 16 central blocks are

counted, thus giving 256 of the small squares, which is sufficient for a fairly accurate average. To count this number of squares after some practice requires about half an hour and the result is generally between 1,200 and 1,500 corpuscles. Much must be learned by practice, and accuracy comes only with experience. After having made the count the instrument should be washed with water and the whole process repeated with a second drop of blood, and in cases of wide variation, a third count should be made.

Enumeration of the Red Blood Corpuscles. The blood is most easily obtained from the ear-lobe, which may be first washed with soap and water. Disinfection is not necessary. Punctures made with a three-sided surgical needle furnish a free flow of blood and obviate the necessity of squeezing, which dilutes the drop. Wipe away the first drops, then draw the blood directly into the capillary tube of the mixing pipette to the 0.5 mark, if a dilution of 1:200 is desired, or to the 1.0 mark for a 1:100 dilution. In cases of anæmia the latter is preferable, while for practice upon normal blood the former suffices and the cells are more quickly counted, with less strain upon the the eyes. Great care is necessary in drawing blood into the tube not to pass the desired mark. In case such accident happens the pipette must be cleansed immediately, and the attempt repeated. The mark reached, the blood adherent to the point of the tube is carefully wiped away and the tube plunged directly into the diluting fluid which is drawn up until the 101 mark above the bulb is reached. The two fluids are intimately mixed by shaking and rotating. The portion which has remained in the capillary tube and not mixed must be expelled, and a portion of the remaining fluid transferred to the counting chamber. The cover-glass is placed over it and the slide is put on the horizontal stage of the microscope where it should be allowed to stand for several minutes in order that the corpuscles may settle evenly. Much care and practice are necessary that neither too much nor too little of the fluid be placed in the chamber. If too much, it will overflow into the rim or moat which surrounds the graduated plate, or overflow between the cell-rim and the cover-glass. If too little, the count will be inaccurate.

If the drop has been accurately gauged, Newton's rings will appear at the edge of the cover-glass as far as it rests on the underlying cell wall, when the glass has been pressed into place.

Diluting Fluids. The simplest is a 0.8 per cent. salt solution, or one of $MgSO_4$ of 20 per cent. strength. Gowers' fluid consists of:

Sodium sulphate	gms.	6.3.
Acetic acid,	gms.	3.6.
Distilled water	gms.	117.0.

Toison's fluid is convenient as it stains the leucocytes and facilitates their enumeration. Its composition is as follows:

Sodium chloride, purif.	1.0	gm.
Sodium sulphate, purif.	8.0	gm.
Neutral glycerine	30.0	c. c.
Methyl violet, 5B	0.025	gm.
Distilled water	160.0	c. c.

Diluting solutions containing mercuric chloride, as Pacini's and Hayem's, are advantageous since they do not stain the tube as does Toison's.

Counting the leucocytes. The same chamber is used for counting the white cells that is used for the red discs. Turck's ruling greatly facilitates the work. For counting the white cells a dilution of 1:100 is sufficiently accurate. If a dilution of 1:200 has been used for the red cells, the second or large bore pipette may be used for this enumeration. The blood being drawn up to the 1.0 mark, Toison's fluid is added until the 101 mark is reached, when the steps described in the case of the red corpuscles are repeated. The leucocytes are stained blue by the fluid and the corpuscles overlying the entire set of large blocks, 144 in number, are counted, beginning at one corner, going across the field and back until all are counted. Corpuscles lying in the interspaces or "alleys" between the blocks must not be included in the count. A rule of procedure must be adopted in regard to counting bodies partially within and partially without the boundaries. Perhaps the safest plan is to count *in* all that lie on two adjacent sides of the square and ignore all that impinge upon the two remaining sides. The rule is equally applicable to the enumeration of both the red and the white cells. Divide the total by 144 to ascertain the average for one square. Since these blocks are composed of 16 small blocks, each of which is 1-4000 cu. mm. in contents, it follows that one of these columns is 16-4000 cu. mm. or 1-250 cu. mm., hence

multiplying the average obtained by 250, by the degree of dilution 100, equals the number of leucocytes per cu. mm. in the blood examined. Thus, if 65 leucocytes are counted on 144 squares, the average per square is $0.45\times250\times100=11.250$ leucocytes in one cubic millimetre of blood. This method has the advantage of being much simpler than that of finding the cubic contents of the microscopic field and should be more accurate.

If for special reasons a dilution of 1:10 or 1:20 be desirable, it becomes necessary to destroy the red bodies, since their preponderance would obscure the white cells. In this case a 0.5 per cent solution of acetic acid to which has been added a little gentian or methyl violet, renders the red cells invisible while it makes prominent the leucocytes.

The apparatus may be cleaned by first washing out the pipette with the diluting fluid, then with water, afterwards with absolute alcohol, lastly with ether. The counting chamber should be washed with water only as alcohol and ether destroy the cement. Explicit directions usually accompany the apparatus.

A magnifying power of three hundred diameters will suffice for the count.

The hæmatocrit, as proposed by Blix and modified by Daland, has the advantages of celerity and ease of management. It consists of two graduated glass tubes, 50 mm. long and 0.5 mm. in diameter, that fit accurately into a carrying frame which revolves in the ordinary centrifuge. The tubes are graduated from 1 to 100. The blood is drawn directly into the tube from the puncture by means of a rubber tube, or it may be diluted with an equal volume of a 2.5 per cent. solution of bi-chromate of potash. For diluting, the pipette of the Thoma-Zeiss apparatus may be used, the blood being drawn to the 1.0 mark, then an equal quantity of the diluent drawn in. The process must be repeated some four times in order to obtain sufficient of the mixed fluid to fill both tubes of the hæmatocrit, to which it may be directly transferred. As a speed of 10,000 revolutions per minute is necessary for good results, an electric current is desirable. The result as indicated by the scale gives the number of corpuscles per cu. mm. by adding five cyphers to the reading, thus, 50 on the scale indicates 5,000,000 red cells. Doubling the scale gives the volume percentage of corpuscles to the normal; thus, 45 on the index equals 90 per cent. If the blood has been diluted, the indicator readings must be multiplied by the degree of dilution.

Much criticism has been bestowed upon the results of Daland's method. It is especially claimed that it is inefficient in cases in which there exists much variation in the size of the corpuscles, or in which the leucocytes are considerably increased; but Daland claims good results in these cases as well as in the others. In a large series of comparative counts made with both the hæmatocrit and the Zeiss instrument, I obtained quite as uniform results as when two different persons counted the corpuscles in separate specimens of blood from the same individual, by means of the Zeiss counter.

Preparation of Dried and Stained Films. Wet preparations can be utilized only when the microscope and proper materials are near at hand. When the examination must be deferred it is necessary to make use of dried specimens. They are prepared by pricking the cleansed finger or ear-lobe, wiping away the first drops, catching the next drop, as it emerges from the puncture, upon a clean cover-glass without touching the glass to the skin, spreading the drop into a capillary layer by covering the one disc with another then carefully sliding them apart, and drying the two films thus obtained in the air. The dry films will keep indefinitely, especially if placed in closed jars.

Simon recommends the use of slides instead of cover-glasses, spreading the drop with the edge of a second slide held at an angle with the first. The superiority of his method rests on the lessened difficulty of gauging the drop to the size of the cover-slip and the more uniform thickness of the smear. My own preference is to bring the edge of the slip even with the edge of the slide and to catch the drop directly in the capillary space thus formed.

Fixation is accomplished by exposing the specimen to a temperature of 140° C. for half a minute, but requires special apparatus. Longer exposure at lower temperatures, as one hour at 110°, will accomplish the same end. Fixation by immersion in a mixture of equal parts of absolute alcohol and ether, or in the alcohol alone, is satisfactory for most purposes. For the first, the immersion should last from one-half to one hour; for the second, five minutes suffices.

The following solution is more satisfactory than either of the above:

Commercial formalin, 40 per cent	1 c. c.
Water	9 c. c.
Mix, add alcohol	80 c. c.

Fix by covering with a few drops of this solution, allow it to remain one minute, then drain off and immediately replace by the staining mixture desired.

With practice one can make perfectly satisfactory stains by simply passing the cover-glass through the flame about twenty times in rapid succession, learned by noting the effect on the red discs. If the heating has been sufficient they will be stained bright yellow by the tri-acid stain.

Ehrlich's tri-acid stain is the most satisfactory for diagnostic purposes and is all that is necessary for the clinician. Gruber's colors are used. The dye is made by mixing the following:

Saturated watery solutions of orange G, methyl green and acid fuchsin are prepared and allowed to stand for several days, after which the clear fluids are decanted. The dye is compounded as follows:

Orange G. sol.	6 c. c.
Acid fuchsin sol.	4 c. c.

Add the one to the other drop by drop, while shaking. When thoroughly incorporated add

Methyl green sol.	6 c. c.
Glycerine	5 c. c.
Absolute alcohol	10 c. c.
Water	15 c. c.

Shake well, let stand for twenty-four hours.

The specimens are stained with the above compound for about five minutes, drained, dried with bibulous paper and examined with the one-twelfth oil immersion lens, with the wide diaphragm.

Ehrlich's triple stain dyes the

Red corpuscles, orange or bright yellow.

Nuclei of leucocytes, green.

Nuclei of nucleated red cells, green.

Neutrophiles, violet.

Mast cells, uncolored.

Eosinophiles, red or copper-red.

The definition of the nuclei is improved by counter-staining with

a saturated solution of methylene blue. After washing off the triple stain the blue solution is poured over the film and washed off after *one* or *two* seconds (Hewes). The blue counter-stain brings out clearly also the malarial parasites, which do not show in the films stained only with the triple stain.

Jenner's stain, the eosinate of methylene blue, gives satisfactory differential pictures. No previous fixing is necessary, as this is accomplished by the methyl alcohol of the stain. The dye consists of a 0.5 per cent. solution of the powder in absolute methyl alcohol. The films are well-covered for five minutes, washed with water, dried and examined.

The red cells are stained terra-cotta.

Nuclei of both red cells and leucocytes, blue.

Neutrophiles, purple-red.

Eosinophiles, bright-red.

Granules of mast cells, dark-violet.

Ehrlich's hæmatoxylin-eosin solution is also known as Gollasch's dye. It is frequently used.

Preparation: Dissolve 2 gm. hæmatoxylin in a mixture of 100 c. c. each of alcohol, glycerine and distilled water. Add 10 gm. glacial acetic acid and a slight excess of alum. The solution must ripen for four to six weeks, after which 0.5 gm. eosin, or, if preferred, orange G., is added. The smears are fixed either by heat or by alcohol (five minutes) and stained for varying times from five minutes to two hours according to the intensity desired. The method requires less skill of technique than either of the others.

The nuclei of the leucocytes are stained dark-blue.

Bodies of leucocytes, light-blue.

Red corpuscles, bright-red.

Eosinophiles, red-granular.

Nuclei of normoblasts and megaloblasts, dark-blue.

Neutrophiles, unstained.

As said, the triple-stain is the only one really *necessary* for the practitioner, and familiarity with one stain and the ready recognition of the bodies differentiated by it is far more useful than doubtful versatility or questionable proficiency.

Malarial Organisms. These parasites are readily recognized in wet specimens by careful focusing, or they may be stained for permanent preparations. The causative factor of malaria is a protozoön

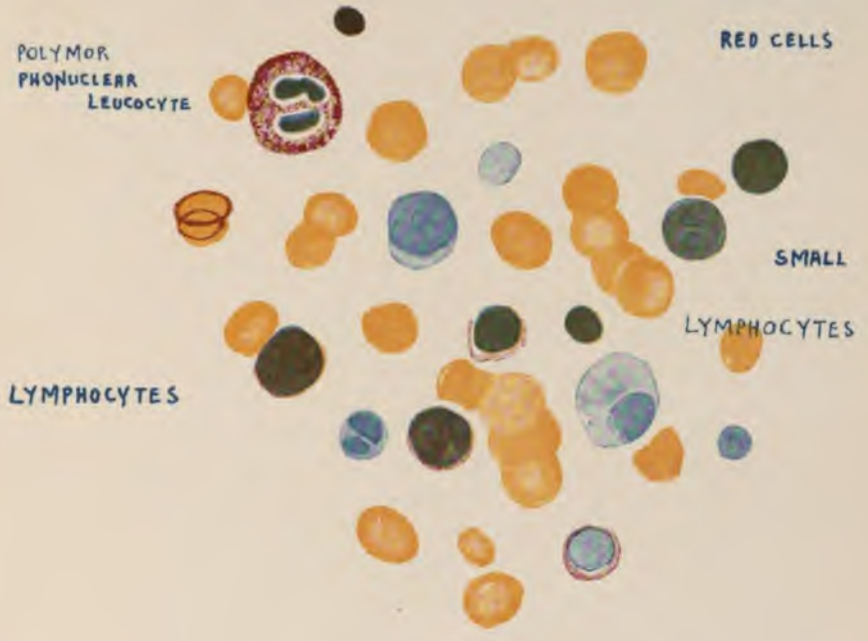


PLATE XIX.

CHRONIC LYMPHÆMIA

Small Lymphocytes in various stages of transition.

of the class which grows in the blood and is therefore called hæmatozoön. This particular species is named in honor of its discoverer, *plasmodium malariæ* of Lavaran. The most common form of the plasmodium is found enclosed in the red blood corpuscle. It is a pale, segmented, mulberry-like body, surrounding a mass of pigment. If a little solution of gentian violet or fuchsin be added to the wet preparation, the stain will impart itself to each of the fifteen or twenty separate segments, differentiating a deep-tinted, central nucleolus from the surrounding protoplasm of lighter stain. Some of these same bodies may be found apparently free from the corpuscle which encapsulated them. The free bodies may be entire or in various stages of disintegration, or exist as small, pale, spherules floating free in the liquor sanguinis, or attached to the outside of the red corpuscle, where they exhibit ameboid movement. Staining here shows also a central nucleolus and a nucleus.

These forms are found previous to, or during, the chill and disappear a few hours after that event, when they are replaced by active ameboid, inter-corpuscular bodies, constantly changing shape and throwing out pseudopodia into the substance of the corpuscle. These undergo successive changes, until the pale spherules again fill the corpuscle, and, at the time of the next chill the rosettes and radiating pigment bodies reappear. The crescents which are found in the blood associated with irregular and severe cases, according to Thayer appear in most of the cases of æstivo-autumnal fever during the second and third week. The crescent develops within the red corpuscle from the small hyaline bodies which gradually increase in size, collect pigment in their centers, assume a crescentic shape, while the outline of the corpuscle disappears. The last to disappear is a line connecting the two horns of the crescent. The dried preparations, after fixing, are best stained by the eosinate of methylene blue, as advised by Romanowsky. The dye may be purchased ready prepared.

Hæmoglobin. The relative values of the hæmoglobin to the corpuscular elements are mentioned in the discussion of the various subjects. It is to be noted that slight alterations in the amount of hæmoglobin are constantly occurring as the result of diet, weather and habits. It is influenced by age, climate, country, sex and disease. The hæmoglobin reaches its lowest value in chlorosis where as low as 15 per cent. has been observed. In this disease the fall in the percentage of hæmoglobin is below the fall in the percentage number of

the red corpuscles, although these also indicate a marked decrease. The corpuscles look pale. In Thayer's series the average for the red bodies was 4,096,000, while the percentage of hæmoglobin for the total number was 42 per cent. In one case the corpuscles were 85 per cent. while the hæmoglobin was only 35 per cent. He calls attention to the fact that there may be, however, well marked actual anæmia.

In typhoid fever the hæmoglobin often diminishes more rapidly than the corpuscles.

In septic infections, especially in general septicæmia, the same phenomena may be observed, but generally the two keep pace with each other.

Blood changes in pernicious anæmia. In progressive pernicious anæmia, the corpuscular loss *exceeds* the loss of hæmoglobin, although the latter may decrease to as low as 20 or 25 per cent. of the normal. The ratio is important as a diagnostic feature. A fall of red corpuscles to half a million is not uncommon and 315,000 has been recorded by F. P. Henry (Anæmia, Phila., 1887). Megalocytes abound; their diameter ranging from 10 to 15 micromillimeters as compared with 6.5 to 9.5 for normal cells. Microcytes and poikilocytes are also abundant in most cases, yet none of these features are pathognomonic.

Minute, highly colored spherical bodies, known as Eichhorst's corpuscles may abound. Eichhorst regarded them as pathognomonic, but they are sometimes absent.

Two kinds of nucleated red corpuscles seen in pernicious anæmia are described by Ehrlich. First, small normal sized corpuscles with deeply stained nuclei. Second, large forms with pale nuclei which he calls gigantoblasts. Leucocytes are usually slightly diminished in number, although the mononuclear white cells may show slight increase. Henry remarks that the red corpuscles in this disease resemble the blood of the lower animals in number, shape and size, and amount of hæmoglobin.

In the latter stages of leukæmia the same ratio as the above is frequently noted.

Blood changes in gastric carcinoma. In carcinoma of the stomach the blood changes ofttimes so nearly coincide with those of pernicious anæmia as to lead to error.

In Osler's series the average of corpuscles in 59 cases was 3,700,000, and the average hæmoglobin in the same series was 45 per cent. The corpuscles varied between 1,000,000 and 6,000,000 while

PLATE XX.

PERNICIOUS ANÆMIA.

the hæmoglobin only fell as low as 30 per cent. The leucocytes exceeded 20,000 per cu. mm. in three cases, and exceeded 12,000 per cu. mm. in eighteen cases.

Blood changes in leukæmia. (a) Spleno-medullary form. In all forms of leukæmia the diagnosis must be made by the examination of the blood, as it alone offers distinctive features. The blood pictures are most striking and significant. In the commoner form, the lieno-myelogenic, the change is the increase in colorless corpuscles. The normal average of white cells per cu. mm. of blood during health has been stated. Their ratio to the red cells is set down as 1 to 500 or 1 to 1,000. In leukæmia the proportion may be 1 to 10 or less. One to 1 is not unknown. The eosinophiles maintain their proper proportion to the other white cells, hence their total increase is very large and their presence in the stained field is striking. Their sizes vary greatly. The polynuclear neutrophiles may maintain normal proportions early in the disease, but frequently diminish in the later stages. The greatest increase is in those cells not normally found in the blood, the myelocytes of Ehrlich. They are marrow cells and are much larger than the large mononuclear leucocytes, from which they further differ by having their protoplasms filled with fine neutrophilic granules. (See classification, 6a.) Eosinophilic myelocytes are also plentiful. (Classif. 6b.)

Mast cells (polynuclear cells with coarse basophilic granules, 5b) are always present in considerable number and may be even more plentiful than the eosinophiles (Osler).

Red cells. The normal red cells are only moderately reduced in spleno-medullary leukæmia, seldom falling below 2,000,000, but normoblasts or nucleated red cells are present, some with large pale nuclei, some showing evidence of mitosis, and gigantoblasts are present. The hæmoglobin value is usually reduced. Ameboid movement is feeble. Charcot's crystals separate out from the leukæmic blood which is kept for a short time.

(b) Lymphatic Leukæmia. As the name indicates in this form the principal changes occur in the lymph apparatus, and in the blood those cells which are derived from the lymph gland predominate, viz: Lymphocytes. The proportion of colorless cells to red cells is less pronounced, seldom if ever reaching 1 to 10; the increase consisting almost solely of lymphocytes, which may be either the small forms or the large cell lymphocytes (2-a) which approach in size the poly-

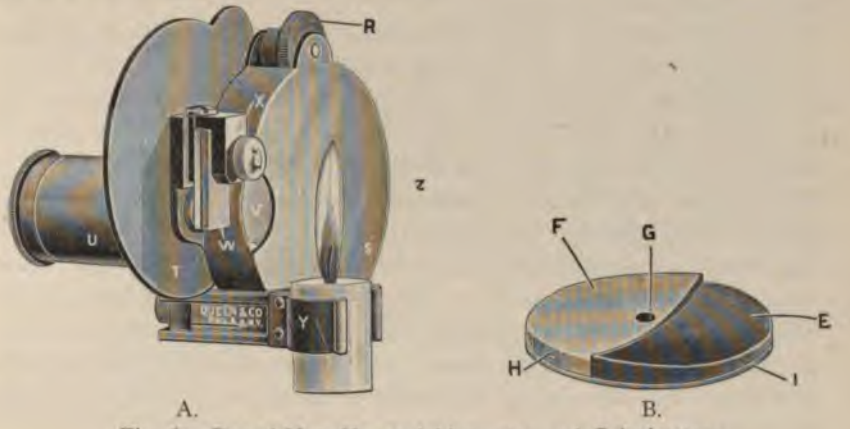
nuclear leucoctyes (3-b). In the purely lymphatic form of the disease, myelocytes are not present and eosinophiles and nucleated red corpuscles are rare, but in the mixed forms, which occasionally occur the blood picture is a union of the two described.

The diagnosis presents only one difficulty; to distinguish it from ordinary leucocytosis. This, however, should present no great obstacle since in all ordinary leucocytoses the increase takes place solely in the polynuclear neutrophilic cells (3-b).

Estimation of Hæmoglobin in Blood. The simplest method of estimating the percentage of hb. is by Tallquists's color scale. (*Nothnagel's Handbuch*, Ehrlich and Lazarus.) This scale is a series of lithographed tints resembling the color of blood containing various percentages of hb. ranging by tens from 100 to 10. The blood-drop is caught on a piece of filter paper and compared directly to the color scale by daylight. Little books of suitable filter paper are sold with the scale. While great accuracy is not attained by this method yet valuable conclusions may be drawn. As normal blood varies between 100 and 90 per cent it cannot be said that a decrease below the normal percentage has occurred until the latter degree has been passed. Tallquist noticed that in stains of blood taken from patients suffering with pernicious anæmia the colored center was surrounded by a zone of varying width, uncolored in some cases, or colored a faint yellow. He observed the same thing in various other anæmias including chlorosis, and concludes that when the nimbus is entirely uncolored, as viewed by transmitted light, the corpuscles have fallen to one-half the normal number or less, and that the greater the decimination, the broader the zone.

A very suitable instrument for the general practitioner for estimating hb. is Gowers' hæmoglobinometer, to be recommended by reason of its low cost and the ease with which it may be used. Results obtained from the better grades of this instrument will probably prove more reliable and accurate to the general worker than would those of the more complicated instruments of Dare, Oliver or von Fleischl, which are better suited to the skilled laboratory technician than to the needs of the general practitioner. The apparatus consists of two small tubes of equal height and diameter, which stand upright in a wooden base. One is closed and contains a colored solution which corresponds in color to a 1 per cent. solution of normal blood in water. The other, open at the top, is graduated into cu. mm. by a number

scale. A lancet, a dropper and a capillary pipette marked at 20 cu. mm., with suction tube, complete the apparatus. A few drops of water are placed in the open tube, the finger is punctured, the blood drawn into the pipette to the mark and immediately discharged into the graduated tube, refilled with water to recover all remaining blood, and this added to the graduate. The tube is now well-shaken and compared with the scale by standing at such an angle that the edges join each other in the line of sight. Water is added little by little, shaking the tube after each addition. The percentage is indicated on the scale when the colors exactly correspond.



A. B.

Fig. 63—Dares' New Hæmoglobinometer and Colorimeter.

Description.

The blood pipette is shown at W. It consists of two small glass plates, one transparent, one white, with a capillary space between, into which the blood to be examined is automatically drawn, undiluted. The plates fit into the detachable clamp, X, and are held evenly in position by the thumbscrew. After filling the capillary tube, the clamp and tube are placed in clamp holder, white glass outwards, as shown in the drawing, where they are held in place by grooved guides in the fixed bracket.

Fig. A shows the color scale. It consists of a prism of colored glass, E, a semicircle of white glass, F, on the edge, H, of which is etched the percentage index of hæmoglobin. G is hole or hub by which the scale is fixed in case, S, allowing rotation. I is a white glass disc which serves as background for the colors.

Fig. B shows instrument ready for use with candle illumination, Y. The color scale has been placed in the metal case, S, and is rotated by milled wheel, R. The light falls equally upon the exposed white glass discs, V and W. The observer views them through the detachable camera tube, U. T, is a movable screen to shade the observer's eye. When the colors exactly correspond, the percentage indicated is read at the scale opening, Z. Th drawings are one-half actual size.

Hodgkin's Disease. The blood shows little that is characteristic apart from simple anæmia of varying grades. The red corpuscles may sink to 2,000,000. They show only moderate poikilocytosis. The white corpuscles may show a moderate increase, with abundant lymphocytes. Occasionally the white cells increase inordinately and resemble the characters found in lymphatic leukæmia.

Purpura. The blood changes are those of anæmia with the hb. line below the corpuscular line.

Addison's Disease. Anæmia is generally present according to Addison, but sometimes is wanting. In Wilson's case the blood resembled pernicious anæmia.

Splenic Anæmia. This is described as a profound anæmia without leucocytosis, and often without marked poikilocytosis.

Pulmonary Tuberculosis. References to the blood changes are included in the general description of the malady. Recent investigations by Ullom and Craig show that when a cavity is present there is a constant mild anæmia, associated with a decrease of the hæmoglobin. They find that an increase of the erythrocytes in cases without cavity is a favorable symptom. A decrease of leucocytes in advanced cases is unfavorable. They regard an actual increase of lymphocytes as indicating an increase in the resistance on the part of the organism against the tuberculous infection, hence such increase as well as augmentation of the transitional forms, is to be regarded as favorable.

SECTION XIII.

DISEASES OF THE ABDOMEN.

The abdominal cavity contains many and varied organs whose physiologic offices, while they differ exceedingly are yet closely related to one another in that they contribute to a common end. Yet in spite of this relationship it may be said that disease of one or the other, whatever may be its nature, gives rise to no symptom-group or correlated phenomena in any way comparable to those associated with disease above the diaphragm. Secretion or excretion may be altered, function may be changed or even abrogated as the result of disease, yet inter-dependent symptoms and inferential physical signs are not a consequence. Perhaps the one exception to this statement is that changes in the blood are apt to be associated with alteration and disease of each of these organs.

For these reasons physical exploration of the abdomen presents many more difficulties and yields less fruitful and less satisfactory results than when the same methods are applied to the thoracic organs. Nor is the reason therefor far to seek. The abdominal organs are many, the thoracic few; the abdominal organs are packed in a loose cavity with distensible walls, and their size constantly changes with their functions. The thoracic walls are almost unyielding the position of the organs is fixed and their capacity but little changed even in severe disease. The contents of the abdominal organs may be solids, liquids or gases, or an admixture of all of these; some may be greatly distended or totally collapsed, as the stomach, intestines, urinary bladder, gall bladder and uterus; while heart and lungs are subject to but little variation. Lastly the action of the thorax and its organs is regular and rhythmical; that of the abdomen and its viscera irregular and intermittent.

The same methods are used which we have seen applied to the thorax but their relative importance changes. Inspection and palpation yield most valuable information here, while auscultation, the most important in thoracic exploration, is of relatively little importance

when applied to the abdomen. The most favorable position for examination of the abdomen is in many cases the erect posture, although recumbency is often necessitated by the form of sickness, or in order to secure complete relaxation. The knee-breast or knee-elbow positions furnishes most information in cases of movable organs, tumors and dropsies. Not only should the front, but also the sides and the back, be examined. Relaxation is secured by elevating the shoulders and gently flexing the thighs. Anæsthesia must sometimes be resorted to.

Anatomy. Whether the segmentation of the abdomen into nine areas has survived the test of time because it is of practical value, or whether it is due to innate conservatism is questionable. Certain it is that the divisions are based neither on substantial anatomic grounds nor warranted by convenience. Variations in the descriptions as found in existing text-books are innumerable and the hope of uniformity is remote. Anderson's suggestion that the boundary lines be converted into planes, defined on the dorsal as well as on the ventral surfaces is here adopted. The plan, however, was originally suggested many years ago by Dr. Bright. The abdominal organs vary widely in their positions within physiologic limits, hence their positions and relations can be described only with approximate correctness.

Two horizontal planes are passed through the abdomen, the upper at the lowest point of the tenth costal cartilage. This plane lies about two inches above the umbilicus, marks the lowest anterior point of the costal border and cuts the second lumbar vertebra posteriorly. It cuts through the transverse colon, the stomach, ascending and descending colon, the lower curvature of the duodenum, the small intestines and the kidneys.

The second plane passes horizontally backward through the anterior superior iliac spines into the pelvis.

A third plane carried across the true pelvis at the upper level of the symphysis cuts the distended bladder, the fundus uteri, the ovaries and Fallopian tubes, the small intestines, the cæcum or lower part of the ascending colon, the sigmoid flexure and the upper end of the rectum.

For the longitudinal planes as pointed out by Anderson the outer borders of the recti muscles—extending from the infra-costal furrow above to the spine of the pubis below—have the advantage of easy location and of giving much greater symmetry to the regions de-

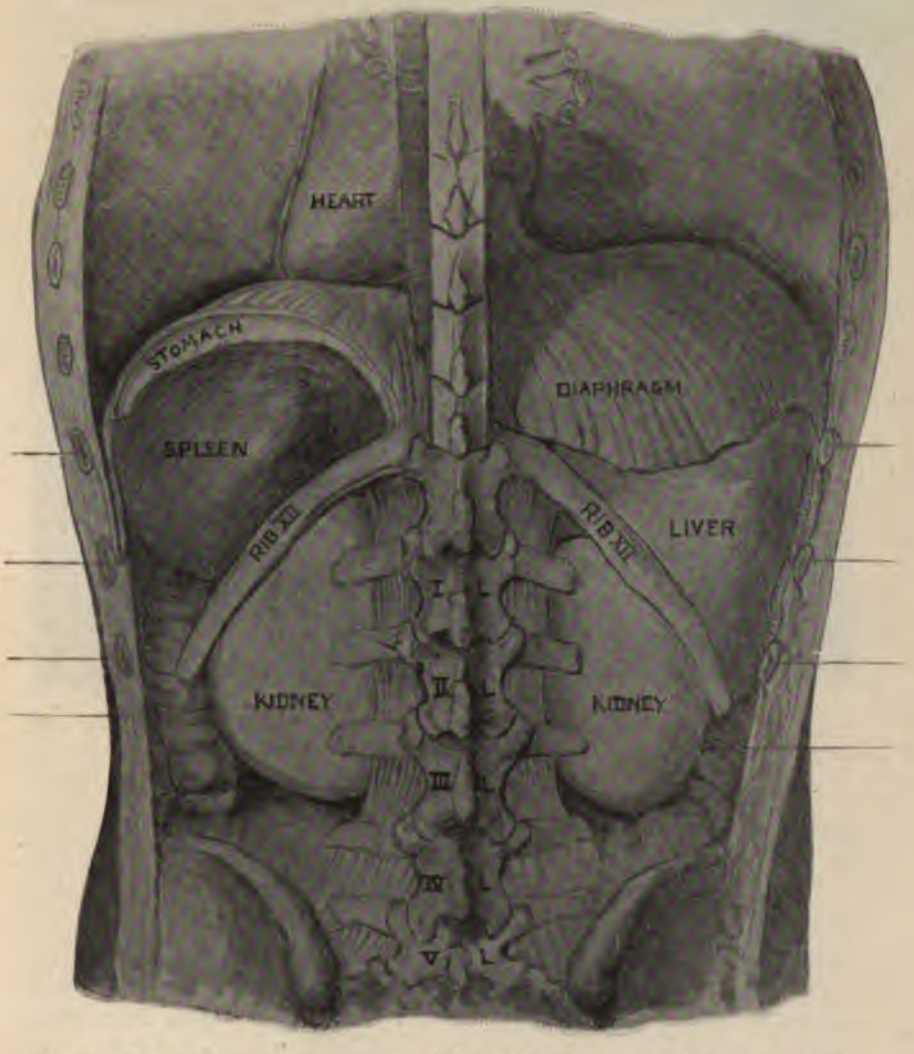


Fig. 64—The abdominal organs viewed from behind. The diaphragm partly removed. From Cunningham's Anatomy, by permission of Wm. Wood & Co.

limited, than the old vertical lines bisecting Poupart's ligaments. The rectal lines, furthermore, bring the inguinal region entirely into one division. The vertical planes cut the transverse colon, the small intestine and kidneys. The ovaries lie at the intersection with the pubic plane. Further, the right plane cuts the gall bladder, the cæcum; the left cuts the stomach, the pancreas, the spleen and the sigmoid flexure.

METHODS OF EXAMINATION.

Inspection. The shape of the ideal abdomen is flatly elliptical with a slight depression marking the umbilicus, a shallow central groove and two vertical lines delineating the inner and outer edges of the recti muscles, and three transverse lines marking the muscle

Fig. 65—A convenient position for palpation of the abdominal organs, which may be used in home or office. Especially useful in searching for small growths of pylorus.

into zones. It is seldom seen except in well-developed, muscular young men. I have never observed the transverse lines in women. They are absent in the very young and those past fifty years.

The size and shape of the abdomen vary greatly in health and its appearance is measurably modified by certain physiologic conditions. The abdomen of the child is considerably larger in proportion to the size of the chest than is that of the adult. Again in old age the abdomen is apt to increase in its volume while the thorax shrinks, thus renewing the disproportion. It is more apt to become voluminous with advancing years in women than in men, and repeated pregnancies leave an indelible stamp. A full meal distends its upper portion.

Probably as an inherited tendency the waist circumference varies more in women than in men. Its disproportion to the girth of the thorax is greater, and in the higher orders of society where lacing has been longer and more strenuously practised the effects are much more marked than in the lower orders, as evinced by our immigrants. It tends to displace the liver and the free end of the stomach downward, as well as to unduly contract the lower ribs which are greatly increased in their inclination, lessening the costal angle and diminishing the lower intercostal spaces.

The pelvic portion of the abdomen is proportionally longer in women and the distance from the xyphoid cartilage to the symphysis pubis varies more than in men, thus giving the female more "waist" and less chest length. We note the size and shape of the abdomen and observe its movements, and look for changes which may result from such alterations. Alterations in the shape of the abdomen due to disease consist in enlargements or retractions of its walls.

Enlargements of the abdomen may be general and symmetrical, or local and irregular. General enlargements are caused by accumulations of gas within the intestinal canal, particularly within the colon (tympanites) as occurs in typhoid fever; by fluid accumulations within the peritoneal cavity (ascites) or in the parietal tissues (œdema); by tumors or new-growths, in which case the enlargement may be local or general.

Local enlargements are in the main due to hypertrophy of some special organ as the liver, the spleen, the kidney or the ovary, to hernias or rarely to enlargements of the mesenteric glands, or accumulations of fæces. Local inflammations such as appendicitis often show local swelling. The descent of the diaphragm and the bulging in the epigastrium due to fluids in the thoracic cavity has been mentioned.

Retraction of the walls is seen in all forms of acute febrile and wasting diseases as typhoid fever, tuberculosis and cancer. It occurs and is progressive in intestinal strictures and in stenosis of the gastric orifices. Infective and catarrhal diseases of the bowels accompanied by diarrhœa, tuberculosis, meningitis and some other cerebral diseases rapidly produce it. It is extreme in lead colic.

Alterations in the movements of the abdomen indicate change in the respiratory rhythm or in the peristalsis. In the unrestricted belly the walls rise and fall with the excursions of the diaphragm. In disease they may increase with the latter or vice versa. Thus in supra-

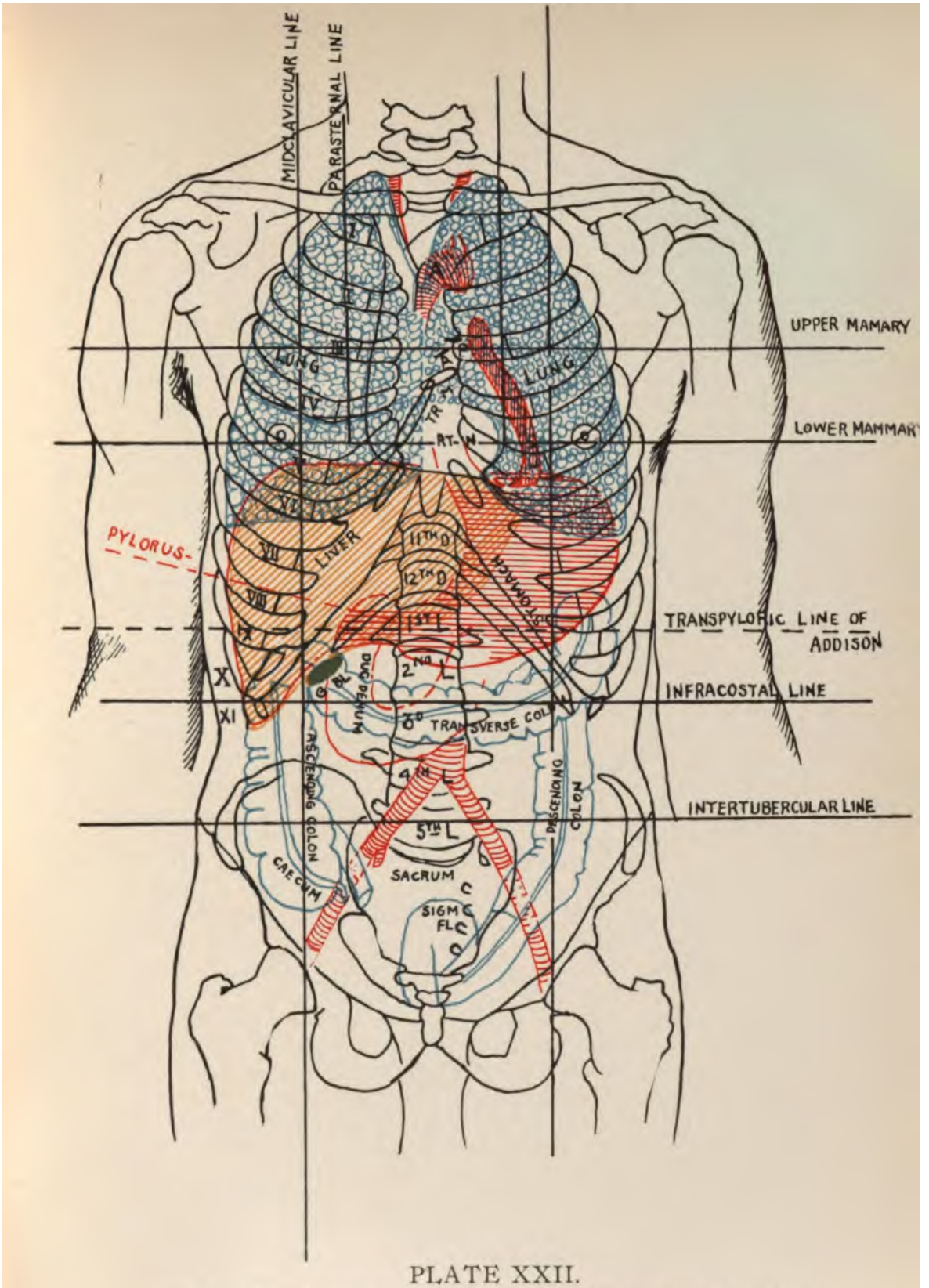


PLATE XXII.

ANTERIOR SURFACE TOPOGRAPHY AND REGIONAL LINES.

A. Aorta.
LV. Left Ventricle.
RV. Right Ventricle.
P. Pulmonary valve.
A' Aortic valve.
M. Mitral valve.
T. Tricuspid valve.
G. Bl. Gall Bladder.

Modified from Cunningham's Ana
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diaphragmatic inflammation, as pleurisy and pericarditis, the muscles are increased in their play, while in inflammations below the partition, as peritonitis or intra-peritoneal growths or colic, their movements are held in abeyance and the costal type of respiration prevails. In thin-walled individuals the outlines of some of the organs and the movements of peristalsis are frequently apparent, and with the loss of adipose in the course of wasting diseases they become so. Semi-rhythmical contractions and relaxations of the abdominal muscles are not unusual, especially in the sick, and the shifting of gaseous accumulation, accompanied by noise, is a common event. The great curvature of a well-inflated stomach may be easily traced.

We examine the state of the skin, whether soft, rough or smooth; moist or dry and branny. Its color, whether it indicates jaundice or impeded circulation. We note distention of veins and capillaries, especially in the umbilical zone already described, and the frequently transmitted epigastric pulsation which is sometimes mistaken for aneurysm.

Palpation. In order to secure the best results from palpation, the complete relaxation of the parietal walls is necessary. To accomplish this, place the patient upon a hard mattress with the legs and thighs partially flexed. It is necessary to change the position from the back to the side, or to the knee-breast posture in detecting the presence of fluids, movable tumors or fluctuation.

The hand should be well-warmed so as not to cause involuntary shrinking and contraction. The side of the hand or the tips of all the fingers used together is preferable to the single finger. Punching and poking are to be avoided. The pressure must vary with the end sought; slight, forcible, continuous or interrupted, being singly or alternately used; care being exercised not to cause useless pain by unnecessary repetition.

Note whether the skin is hot, indicating fever or inflammation, or cold, indicating chill or deficient circulation; whether the tissues are unusually relaxed or resisting and elastic and whether any œdema exists.

The examination of the contained organs determines their size and position, whether fixed or movable and whether pulsation is present; their consistency and outline, whether smooth, nodular, tense, tender, softened or hardened.

Fremitus due to enlarged spleen or liver may occasionally be detected.

Fluctuation means the presence of fluid. In order to detect it the examiner places one hand at the distal part of the collection, and taps upon the overlying surface. The peculiar stroke communicated to the hand by the fluid set into vibration is called *fluctuation*. Circumscribed fluctuation is distinguished from fluctuation due to fluids free in the cavity by its area and position and by not gravitating freely with changes in the posture of the patient. Such collections occur in ovarian cysts, ligamentous cysts, hydatids of the liver and possibly in cystic kidney. Encysted collections of pus or other fluids limited by peritoneal adhesions, fluids distending the fallopian tubes, and abscess of the abdominal walls, may also give rise to localized fluctuation.

Pain may be superficial or deep and the amount and kind of pressure necessary to produce it determine this fact. The character of the pain and the manner in which it is influenced by palpation have no little significance. Pains due to inflammation of the bowels or other mucous membranes are dull, heavy and continuous and require considerable pressure to manifest them. Pain arising from inflammation of the serous coverings, the peritoneum, is sharp and cutting, subject to exacerbations and increased by slight pressure. Colicky pains, nerve pains or neuralgias are often relieved by firm pressure, although a touch may cause a painful spasm, which distinguishes them from the tenderness resulting from inflammatory troubles.

Palpation includes vaginal and rectal examinations, for fuller details upon which subjects the reader is referred to special works.

Percussion. By percussion we determine the size and position of the various organs, their relations to each other and even gain considerable information as to their contents, since the note varies unmistakably therewith. The position of the patient best suited for percussion is recumbency upon the back, as for palpation. The fingers furnish the best plexor and pleximeter, and the variations in the note elicited are so manifest and so readily recognized that the student will do well to familiarize himself with their qualities before essaying thoracic adventures. These notes are dull, flat and tympanitic according to the point chosen and the organ percussed. The body of the liver, the spleen and the kidneys give forth a perfectly flat note,

while their edges which overlap the stomach, colon or intestines give a varying dull note. The small intestines, the colon and the stomach give a full tympanitic note, which may be characterized as small, medium and large for the three viscera in the order named. The separation of the three organs nevertheless requires considerable delicacy of discrimination, by reason of the variations of the note which arise from different conditions of these viscera. Thus, each may be distended to its fullest capacity by gaseous or solid contents, or may be in a state of collapse, or they may be partly filled. In each condition the note for the same organ will differ. The quality of the note varies but little, being tympanitic in all cases except when the viscus is filled with solid matter, but the pitch varies widely. As stated in our preliminary considerations, pitch rises with the tension but diminishes as the enclosed air volume increases, *provided the tension remains the same.*

From the foregoing it will be seen that the note of large tympany, or stomach tympany gotten by percussing a moderately distended stomach which is normally the lowest pitched sound obtainable from the human body, rises in pitch if the gas so distends the stomach as to increase its tension, when the note may become metallic or amphoric.

The intestines, being smaller cavities, give a higher pitched note than the stomach; the tension being equal, this note is higher for the small intestine than for the colon.

Increase in size of the solid organs, as well as new solid growths, are recognizable by the extension of their area of flatness; thus, enlarged liver, kidneys, spleen, uterus, tumors of ovary, womb or mesenteric glands or aneurysm of the great vessel may be recognized and their limits determined. A full bladder gives us an area of dullness above the symphysis, where intestinal tympany replaces it when emptied. So too, distentions of the hollow viscera manifest themselves by a change in the percussion note, the tympany increasing with the distention.

It will thus be seen that intelligent percussion furnishes valuable results. In order to obtain the full benefits the areas must be carefully gone over from side to side and from above downward with a stroke of varying strength; now light, to elicit superficial resonance; now medium, to determine relative dullness; and strong, to demonstrate deep-seated, covered-in flatness.

The percussion areas of the various organs will be given in detail along with the consideration of their diseases.

The state of the abdominal walls may interfere with the satisfactory examination of the organs: thus accumulation of fat seriously influences palpation and the percussion note. Contraction and spasm of the parietal muscles, as occurs in beginning peritonitis, or the unilateral contraction of the rectus as seen in appendicitis, may retard the examination yet contribute to its diagnosis. Œdema of the walls, such as occurs in chronic Bright's disease, seriously interferes with the examination.

Auscultation. Over the abdominal tract no sounds are heard in health save the whirr of the blood through the aorta, the shifting of gas in the intestines known as "borborygmi" and certain churning sounds of a somewhat metallic character which arise from the mixed contents, solids, liquids and gases, of the stomach.

In disease the bruit of an aneurysm may be present. A friction fremitus due to two inflamed surfaces of peritoneum rubbing together is much rarer than friction sounds in the region above. A blowing thrill or murmur is sometimes heard over the spleen when that organ is enlarged.

The placental bruit and the sounds of the fœtal heart belong more properly to the field of obstetrics, but are pathognomonic of the pregnant state.

DISEASES OF THE PERITONEUM.

The peritoneum lines the entire abdominal cavity and is so reflected as to enclose wholly or partly all the contained viscera, vessels and structures.

PERITONITIS. INFLAMMATION OF THE PERITONEUM.

Classification: Acute, chronic; primary, secondary; local, general; simple, septic.

Morbid varieties: Fibrinous, serous, purulent.

Symptoms: Pain; severe, cutting, piercing, griping. In puerperal cases less severe. Tenderness. Seat of pain and seat of tenderness may not coincide. Chill; vomiting, early and severe. Vomited matter; food, bile and even fæces.

Fever: Generally 101° to 103°; evening rise.

Pulse: Rapid, increases with disease, 130 to 140.

Respirations: Superficial, painful, shallow. May be 30 to 40 per minute.

Physical signs: Patient lies on the back with the knees flexed. Abdominal walls at first tense and retracted, resistant. Diminution or suppression of abdominal breathing; thoracic breathing increased. Later abdomen swells and becomes tympanitic, resistance passes.

Disappearance of liver dullness points to presence of gas free in the peritoneal cavity, but previous adhesions may hold the liver against the parietal wall or a coil of distended intestine may intervene and replace the dullness. Pain is manifest on slight touch, but actual seat of tenderness is only determined by firm pressure. Fluid, indicated by fluctuation, gravitates into the cavity while the intestines float on top. Dull area shifts with the changes of position. Hiccough may indicate involvement of the diaphragm covering. Friction or crepitation is often heard. Heart is displaced upwards or outwards, apex may be in the fourth interspace and heart and liver dullness be found in the third interspace.

Urine scanty, dark, heavy, contains albumin and indican in large quantity. Simple test: Shake together equal parts of HCl and urine, then add three drops of a freshly-made, saturated solution of chlorinated lime. Indican is indicated by the dark blue color which immediately succeeds.

In the chronic variety the chief manifestation is a collection of fluid in the peritoneal cavity. The collection occurs independently of tuberculosis, cancer and causes of ascites.

THE STOMACH.

When the stomach is empty, or not distended with gas, its outline and position are not indicated by any prominences on the outer wall, and palpation as well as inspection fails to furnish any information as to its location. Since, however, the viscus is seldom if ever in a state of collapse, percussion gives us a tympanitic resonance so distinctive and drum-like as to enable us to differentiate it from the neighboring organs. The flatness of the percussion note which marks the liver area limits the stomach above and easily determines its upper boundary. The inner border of the spleen limits the fundus on the left. The degree of obliquity of the stomach is still a matter of dispute, the merits of which we will ignore at present, stating what we consider the most accurate views. When the stomach is distended

the fundus and greater curvature rotate to the front, and come in immediate contact with the abdominal wall. This portion is triangular in shape, bounded on the right and above by the liver, on the left by the costal edge as low as the ninth. The free border or greater curvature can be determined by percussion. Begin above at the edge of the liver and percuss gently downwards from this line of dullness until a slight alteration in the pitch of the tympanitic percussion note indicates that the transverse colon has been reached. This line, which marks the greater curvature of the stomach, bows downward from the free end of the tenth rib on the left, crosses the middle line a little

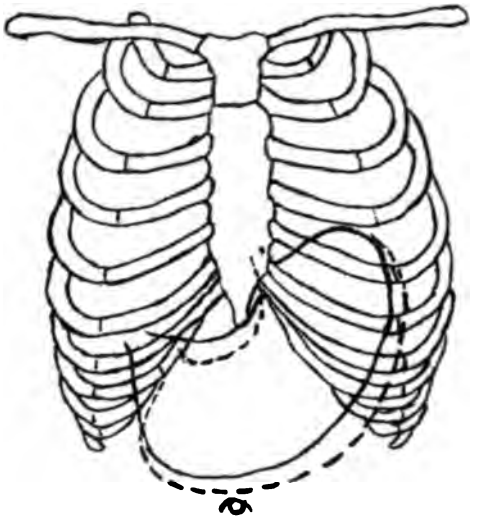

Fig. 66—Normal position of the stomach, according to Rosenheim (solid line). Dotted line, normal position according to Eichhorst.

above the umbilicus (1″ to 2″), thence curves upward to the edge of the thorax where it meets the right parasternal line.

The cardiac end or fundus reaches as high as the sixth cartilage being behind the heart apex. The cardiac orifice is opposite the seventh left costal cartilage about one inch from the sternum (Treves).

The pylorus varies, but its most frequent position is to the right of the middle line two to three inches below the sterno-xyphoid articulation. This point corresponds with the level of the twelfth thoracic spine.

For diagnostic purposes the stomach may be distended either by inflating it with air or by means of an effervescing powder. Air may be introduced through the stomach tube by means of a hand bulb. The powder consists of a teaspoonful of tartaric acid dissolved in a half-tumberful of water and swallowed, to be immediately followed by a teaspoonful of sodium bi-carbonate also dissolved in a half-tumblerful of water and the gas retained. The resulting distention is usually visible and the outline may be thus determined.

Knapp describes a simple method for locating the lower border of the stomach, which I have found useful.

The patient, standing erect with the abdomen covered, drinks a glass of cold water. After the lapse of half a minute the examiner places his hand, which must not be cold, against the bare abdomen. The lower limit of the cold region is quite definite, and denotes the position of the fundus and greater curvature of the organ.

Lincoln, by a method of transillumination of the stomach by the use of fluorescin, gives results bearing upon the position of the lower border of the viscus in thirty cases. In four he found it above the umbilicus, in four at the level of the umbilicus, in twenty-two below that level. He leads us to infer that only eleven of those examined had gastroptosis; and concludes that the stomach lies much lower in the healthy individual, male and female, than generally has been described.

Enlargements of the stomach may be recognized by palpation and percussion; diminution in its size cannot be told by physical exploration.

Increase in the size of the stomach may be temporary or permanent. Temporary distentions are of daily occurrence and cannot be regarded as pathologic. They are caused by accumulations of gas or the ingestion of large quantities of liquids or solids. Treves gives the average capacity of the stomach as about five pints (two to three litres). This is certainly too great, three points (1½ litres) being more nearly correct. The elder Leidy used to say that stomachs which have never been over-distended were filled by a pint but could be stretched to two quarts (two litres).

Gaseous distention of the stomach is recognized by the note of large tympany heard over an extended area. It should not be difficult to separate this sound from the note of the colon, unless the latter is also greatly distended. When the stomach is distended by

liquid or solid contents the normal tympanitic note is wanting, and the dull area which replaces it will extend from the liver edge down to the umbilicus, and outward on the left until it fuses with the splenic dullness. When the stomach is partly filled, as some time after a meal, the contents gravitate to the dependent part or cul-de-sac and the dullness is limited to that portion of the organ. For the purpose of detecting these conditions percussion should be made with

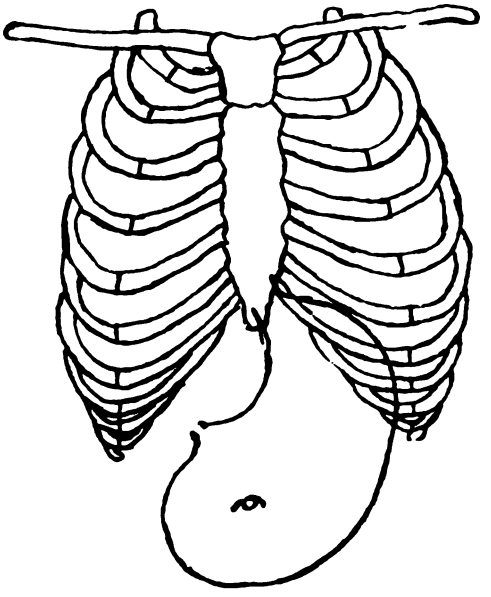

Fig. 67—Position of stomach in moderate gastroptosis (Rosenheim's description).

the patient standing. In case the viscus is empty this is sufficient, but if partially filled the percussion should first be made with the patient erect, noting the dull area, then repeated with the patient lying upon his back, when the dull area will be replaced by the characteristic tympany.

Almost all forms of dyspepsia and conditions in which stasis of the gastric contents occurs are associated with gas formation and temporary distention. The stomachs of beer-drinkers are apt to be distended. The increase in size may be permanent, as in gastrectasia, which is a permanent increase in the volume and capacity of the stomach, arising from muscular atony, or in pyloric obstruction, in

which the condition is brought about by the gradual stretching of the walls by the retained contents.

Physical signs of Gastrectasia. Inspection may reveal the outlines of the organ, especially the greater curvature, which extends as low as the navel and even lower. As these cases are very generally accompanied by descent of the organ, the upper curvature may sometimes show itself circling beneath the ensiform cartilage.

Palpation. The air cushion sensation is often recognizable by the touch.

Percussion furnishes trustworthy evidences of dilatation. We begin above and percuss downward to the colon, observing the precautions mentioned as to the change of position of the patient. If the viscus is empty the ingestion of a large glass of water will cause the lower tympanitic border to be replaced by dullness when the patient is erect. The double percussion is conclusive. If doubt remains a stiff sound may be passed and the end felt through the abdominal wall, or the sound may be pushed along the great curvature until it encounters the resistance of the pylorus and the abnormal distance noted. If the lower limit of the stomach reaches below the navel the conclusion that the organ is dilated is justifiable. The stomach tube may be used for filling the organ with water in order to displace the tympanitic note, but is seldom necessary.

CANCER OF THE STOMACH.

One in five of all primary cancers attacks the stomach, about two-thirds of the cases attack the pylorus.

Inspection. In the early stages, negative. After cachexia and emaciation set in the tumor may be visible.

Palpation. Tenderness may be present long before a tumor can be felt. When palpable, tumor of the pylorus is generally situated near the umbilicus, owing to the displacement caused by dilatation, and is freely movable, changing its position with varying conditions of the organ and the body.

Percussion gives an area of dullness—not flatness—over the tumor wherever it may be situated. The percussion should be performed when the stomach is empty or has been washed out, when the contrast of tympany and muffled dullness is more manifest. Pyloric stenosis, which results in most cases, causes gastric dilatation with the physical

signs of that condition. Sometimes peristalsis, stopping short at the pylorus, may be seen.

Other symptoms are those of dyspepsia and debility; vomit which contains blood ("coffee grounds"); diminished or absence of HCl and the presence of lactic acid in the stomach contents.

DISEASES OF THE SMALL INTESTINE.

Anatomy. The small intestine extends from the pylorus to its junction with the large intestine in the right iliac fossa and has an average length of seven meters. It is divided into the duodenum, 30 centimeters long; three-fifths of the remainder constitutes the jejunum, the balance forming the ileum. There is little histologic distinction between the two structures. Two kinds of glands are found in the walls, secretory—the glands of Brunner and Lieberkühn—and the agminated glands or Peyer's patches and the solitary follicles.

Peristalsis. During digestion the gut is subject to certain movements designed to propel forward its contents, called peristalsis. These movements are wave-like—peristalsis proper—pendular and rolling. Violent painful peristalsis is called tormina.

The functions of the intestines are absorption and excretion. A considerable quantity of gas is always found within the lumen, derived from the stomach, the blood or formed within the canal (see Fæces).

Previous diseases, habits, occupation, diet and the use of special substances as alcohol, tobacco or drugs should be the subject of careful inquiry in intestinal affections.

The principal facts to be elicited are as to the existence of

(a) Pain; its seat, character, duration, relation to digestion, evacuations, eructations and food ingestion; how influenced by rest, motion or pressure.

(b) Tympanites. Sudden tympanites should suggest obstruction, especially if inability to pass gas co-exists.

(c) Constipation, its duration and nature.

(d) Diarrhœa, the character of the stools (see Section XVI).

(e) Tenesmus. This usually indicates an affection of the large intestine.

The maladies of the small intestine individually considered do not give rise to definite correlated groups of distinctive physical signs, hence these manifestations may be considered together, afterwards

pointing out the differential characters along with the special symptoms.

Inspection is not limited to the abdomen, but the chest, the back and the anal region should be carefully viewed. The examination is best made upon a hard bed or couch with as perfect relaxation as possible. The import of various superficial vascular conditions is referred to elsewhere. Irregularities of contour, the condition of the skin, the presence of ascites or œdema are especially to be noted. Ewalt says small new-growths are often easier to recognize by inspection than by palpation. Wasting diseases, cancer, the cachexias, lead-colic and meningitis give rise to depressed or scaphoid abdomen. The contour and outline should be inspected with the eyes on a level with the surface, viewed during full inspiration and forced expiration. Irregularities are sometimes revealed by voluntary muscular contraction of the walls.

In nervous and emaciated persons, particularly the convalescent, and in those with relaxed abdominal walls peristalsis is visible. If general and confined to the small intestine it is not especially significant. Stopping short at a given point it indicates obstruction. Visible peristalsis of the large intestine is pathologic and not infrequently points to stenosis. It is also found in neurotics. During attacks of colic sudden forceful peristaltic discharges—tormina—(accompanied by shifting gaseous contents) are often visible.

Palpation. The desirability of a warm room and warm hands has been mentioned. Palpate first the superficial parts, then the deeper regions, examining first with the patient on the back, then on either side, lastly in the knee-elbow position. A number of writers advise palpation in a warm bath.

After a careful examination of the parietes, the deep palpation best proceeds from above downward, not omitting the inguinal and femoral canals. The course of the large intestine should be separately followed. The sigmoid flexure is easily palpated. An increasing number of observers, since Edebohls, claim ability to palpate the appendix—certainly a difficult task.

We note by palpation irregularities of distention, pain, sensitiveness, tenderness, succussion, adhesions, new-growths, fæcal accumulations. As to the last, differentiation has been sufficiently dwelt on.

New-growths are much oftener malignant than benign. The size, mobility, consistency, contour and location are noteworthy.

Cancer, sarcoma and tubercular growths are the most frequent. The first are irregular, hard, nodular and seldom attain large size. The second smooth, hard and may be large.

The third vary in consistency, are diffused, occur in young subjects and are often associated with tuberculosis elsewhere. Diarrhœa accompanies these while the former are associated with constipation.

Percussion. General directions upon abdominal percussion and the results obtained by percussing different areas have been given. We search for departures from the normal note. Successive examinations, especially after freely evacuating the bowels, are recommended. The character and differences in the sounds elicited have been described.

Auscultation reveals intestinal sounds such as succussion, borborygmi and gurglings. Little of practical value is educed from their presence. Their absence is significant of paralysis of the bowel, as in appendicitis, or of perforation, in which case there is total absence of all bowel sounds. In stenosis the sounds are increased. Gurgling in the right iliac fossa, formerly considered a diagnostic feature of typhoid fever, has no clinical value.

ULCER OF THE DUODENUM.

These occur at any age, even in the new-born, but are most frequent between the ages of thirty and fifty. Men are more frequently affected than women. The symptoms are similar to those of gastric ulcer, for which the disease is apt to be mistaken. The special symptom is pain which appears several hours after eating. The situation of this pain is on the right parasternal line just below the gall bladder. It has been found that diminishing the acidity of the gastric juice or temporarily suppressing its flow through the pylorus relieves this pain, a feature which distinguishes the disease from gastric ulcer. Administering acid wines or food accomplishes the purpose. Tenderness, reflex vomiting, hæmatemesis and blood in the stools summarize the symptoms. The gastric contents give no definite indications. Perforation or fatal hæmorrhage may occur.

ACUTE AND CHRONIC INTESTINAL CATARRH.

These may be classed as infectious, alimentary, climatic, medicinal and toxic catarrhs. The first is primary or a complication of existing disease; the second includes the larger majority of all cases.

The general symptoms are pain, nausea, diarrhœa with much or little mucus. The presence of green bile in the dejecta indicates that the disease is in the upper intestine. General lassitude and weakness supervene. The stomach is often involved. Fever is slight or absent. The urine is scanty and not seldom albuminous.

The diagnosis is easy. Large masses of mucus and an admixture of blood with the fæces point to the large intestine as the seat of the disease. Local tenderness aids in locating it. The main symptom is the altered condition of the fæces.

The symptoms of the chronic form are similar but less acute. Constipation *is not incompatible with this form*, a fact to be remembered. Constipation and diarrhœa may alternate in chronic catarrh.

Membranous colitis is a form of catarrh accompanied by painful spasm of the intestine, alteration of function and the occurrence in the stools of mucus which assumes the form of membrane or casts. Females are usually the subjects of the disease and constipation marks the preponderance of the cases.

Intestinal obstruction or stenosis may affect the small or the large bowel. The causes are formation of ligamentous bands, the occurrence of hernia, impaction by gall stones, foreign bodies (food, fæcal impactions), intestinal concretions, compression from organs or growths and intussusception. Band or slit strangulation and intussusception account for ninety per cent. of the cases. Dilatation develops above the obstruction and is greater the nearer the difficulty is to the rectum. For this reason when the jejunum is the seat, involvement and over-distention of the stomach quickly intervene. Constipation comes on as soon as obstruction is complete but may have been preceded by diarrhœa. The peristalsis is augmented and is visible, occurring in intermittent painful spasms. The character of the stool is not significant. In certain cases bile is constantly found in the stomach. The subjective symptoms are loss of appetite and strength, nausea, fullness and a peculiar distress. Eructations and vomiting mark most cases. Visible peristalsis and intestinal rigidity are the most important physical signs. Suddenly developed constipation, resistant to cathartics, occurring in one of regular habits should excite grave suspicion, particularly if the patient is of advanced age.

Complete, rapidly-developed obstruction involving one or more intestinal coils, is called *ileus*. The symptoms are great pain, nausea, vomiting and suppression of fæces and gas. The vomiting if con-

tinued becomes stercoraceous. Incarcerated hernia is the most frequent and potent cause. Tympanites develops, but spasmodic peristalsis is generally absent. The distended and resistant portion of the intestine before the obstruction can generally be recognized by palpation and inspection. In hernia, palpation recognizes the incarcerated bowel by the impulse communicated on coughing.

Obstruction may be mistaken for biliary or renal colic, but the history, the presence of jaundice, enlarged, tender liver or kidney, bile, blood or other foreign substance in the urine aid in recognizing these while marked indicanuria points to obstruction. The symptoms tending to exclude peritonitis are given thereunder.

Volvulus is a twist of the intestine upon its mesentery, or a knotting of two or more intestinal coils. The sigmoid flexure is oftenest involved, next the ascending colon.

As a rule it is an accident of late or middle life.

Pain, absence of fæces and flatus, local tympanites and vomiting are present. Tenesmus is inconstant. The fæces below the twist, when evacuated, may contain blood. Constipation resistant to cathartics follows.

Palpation. Sometimes the flexion can be palpated, or we may discover in the lower abdomen near the median line a resistant, air-cushion tumor of varying size.

Percussion evolves dullness or tympany according as the sigmoid is full or empty. Metallic tinkling is occasionally heard. The immediate return of water injected into the rectum and the ability to inject only small quantities aid in the diagnosis.

Intussusception is the invagination of one portion of the intestine within the lumen of another portion. It may occur at any age, but is especially prone to affect infants and young children, and appears suddenly.

Symptoms. Spasmodic pain, tenesmus, blood in the stools, which continue until the distal portion of the bowel is evacuated, and the presence of a palpable tumor, hard and sausage-like, over the ascending or transverse colon make certain the diagnosis. Blood in the stool is found in over eighty per cent. of the cases. Intussusception occurring during the death agony is not to be mistaken, when found post-mortem, for the above.

GALL STONE DISEASE.

Synonyms: Cholelithiasis, Hepatic Colic, Lithogenous Biliary Catarrh.

Definition. An acute or chronic affection of the gall bladder and ducts, generally inflammatory, resulting in the formation of calculi.

Bacterial invasion is a potent, if not the sole, cause.

The sufferers are generally women who have borne children; persons between the ages of thirty-five and fifty. In many cases stones exist without causing inconvenience. Stones are single and large, or multiple and small. Permanent obstruction of the cystic duct results in dropsical enlargement of the bladder—*hydrops vesicæ felleæ*—which results in a cystic tumor discernible in the upper abdomen, and easily recognized.

The general symptoms of the colic are produced by the passage of the stone through the duct or by its lodgment therein. The former is the cause of ordinary biliary colic.

Symptoms. Pain, severe, even agonizing, which comes on suddenly. Generally centering in the right hypochondriac region it radiates over the epigastrium, lower thoracic region and into the right shoulder and arm.

Palpation. Tenderness which centers over the gall bladder is manifest and the bladder may be palpable. With relaxed abdominal walls gall stone crepitus is occasionally felt when the bladder is not over-filled with stones and its walls are not too tense. Occasionally the muscles of the right side are rigid. Enlargement and tenderness of the liver, and enlargement of the spleen, are discoverable by palpation and percussion. When the stone reaches and blockades the common duct it produces jaundice, which may be slight and transient, or intense and protracted. The colic lasts hours or days, ending suddenly with the discharge of the calculus into the bowel. (For recovery and recognition of gall stones, see Section XVI.) Rigor and chill are occasional. Fever is generally present; temperatures of 102°—103° are common, 105° is exceptional. Vomiting and protracted nausea are usual. The urine contains bile when jaundice occurs, and bile in the urine frequently precedes its manifestation in other localities. Albumin and red blood corpuscles are found in most cases.

Differential Diagnosis. The location of the excruciating pain and tenderness are almost sufficient. If jaundice occurs the diagnosis is sure. Stone should always be sought for in the stools. History of previous attacks greatly aids.

From renal colic it differs in the location and character of the pain. There the pain is in the lower abdomen, radiates to groin and testicle; icterus is not present, nor is bile found in the urine. Gastralgia is without chill, fever or jaundice: vomiting is rare. Pressure and food ingestion afford relief. The attacks are periodical and associated with general nervous manifestations.

APPENDICITIS.

Definition. An inflammation of the vermiform appendix caused by the invasion of micro-organisms. The disease may be acute or chronic.

The recognized varieties are: Catarrhal, Interstitial, Ulcerative, Gangrenous.

The effects of extension of the inflammation beyond its original bounds are circumscribed or diffuse abscess, and peritonitis which may be limited or general. These results are brought about by extension of inflammation, by perforation or by gangrene.

Catarrhal appendicitis may result in immunity from further attacks by adhesion of the contiguous surfaces producing obliteration of the lumen—a fortunate but rare result. Obliteration of the proximal portion may result in abscess or cyst of the distal part. Foreign bodies are occasionally found in the appendix, fæcal concretions frequently, but their influence in producing disease has been over-estimated. The possibility of tubercular and typhoid ulceration of the appendix must be kept in mind.

Children and young adult males are the most frequent subjects of attack.

Symptoms and physical signs. The lighter grades of the disease, especially the catarrhal variety, undoubtedly often pass unnoticed or occasion only slight pain, passing fever and some disorder of the bowels. The interstitial form gives rise to more definite symptoms, occasioned by involvement of the peritoneal coat. The gangrenous and ulcerative forms may be insidious but usually give rise to definite symptoms. The oncome of gangrene sometimes lulls the pain and masks the pre-existing signs.

The first, most constant and invariable sign of appendicitis is *pain*, paroxysmal or colicky. In the beginning this initial pain is intermittent or remittent, variable as to seat and severity. Oftenest centering around the umbilicus or over the epigastrium, less often in the right iliac fossa, it finally settles over the seat of the appendix after a time varying between twelve and thirty-six hours. If the organ is in an anomalous position the seat of pain will be likewise. The general location is now in the fossa at a point on a line drawn from the umbilicus to the right anterior superior iliac spine, distant one and one-half to two inches therefrom. This point is external to the rectus muscle and is known as McBurney's point. The pain is called the secondary pain.

Allied to pain is *tenderness*, the second cardinal symptom. This may be limited to a rather small area surrounding the underlying inflammatory focus or may extend over the entire right half of the abdomen. Not infrequently the lower right quadrant limits the tenderness. When the entire belly is affected it points to an extension of inflammation beyond the original focus. The seat of maximum tenderness and maximum pain usually coincide and correspond to the point named above, but the pain may subside and the tenderness remain. In case of suppuration the tenderness becomes exquisite.

As first pointed out by Deaver, the third most significant symptom of appendicitis is *rigidity of the muscles of the right side of the abdomen*, particularly the right rectus. This rigidity comes on early and lasts throughout the primary stage of the disease, or until suppuration, abscess, peritonitis or declination of the inflammation succeeds.

In case the disease progresses unfavorably the pain, tenderness and muscular rigidity may subside, or are wholly or partly replaced after a varying time, generally two or three days, by a localized swelling, or by a general distention of the abdomen. The local swelling varies in size and consistency but is usually above the size of a small lemon, lying well within the iliac fossa above Poupart's ligament.

Peritonitis resulting from suppuration does not differ in its manifestations from peritonitis herein described due to other causes.

Vomiting is a general and early symptom. While usually ceasing with the evacuation of the stomach, occasionally bile and even fæcal matter are vomited. Continued vomiting is an unfavorable omen, as is likewise hiccough.

Fever is always present. Although in the early stages its severity can not be accepted as an index of the gravity of the attack, yet in general its decline is a favorable symptom. Suppuration is accompanied by a maintained or rising temperature. The characteristic pus curve rarely is present. Suppuration may occur after the decline of the fever, as may also gangrene. Continued or increased tenderness is a valuable sign of these conditions.

A sudden fall of temperature, cold bedewed skin, small pulse, in short the signs of collapse, indicate perforation. It is to be remembered that the severity of the disease may increase without any thermometric warnings. Even afebrile cases are said to occur.

The *pulse* varies with the fever; at first rapid and full, with unfavorable progress of the disease it becomes rapid, small, thready and compressible. Deaver considers the pulse changes a more reliable index to the disease than the temperature chart.

The bowels are usually constipated at the outset and the malady very rarely succeeds upon an attack of diarrhœa, but diarrhœa may intervene after the beginning of the attack, and, on the whole, in my own experience is to be considered a favorable symptom. Alternate diarrhœa and constipation may occur during the disease.

The urine is febrile, may contain albumin and casts. As mentioned hereafter the presence of indican is indicative of suppuration.

Urobilin is often present.

The blood changes are given in Section XII.

Physical Signs. Inspection in most cases is negative. The patient usually lies upon the back with the right leg flexed. The expression indicates pain. In advanced cases the look is anxious. At first there is slight retraction and fixedness of the right side. If tumor forms swelling is observable but seldom reaches large proportions. It may extend backward to the flank. The resultant paralysis of the bowel may cause local or general distention of the abdomen. Peritonitis is accompanied by rapid distention of the abdomen and extension of the swelling. With the peritoneal involvement the respiratory rhythm is interrupted, the abdomen restrained and the breathing becomes costal.

Palpation. The difference in the degree of resistance offered by the two recti muscles is marked and continues throughout the first stage of the malady. On deep palpation an area of resistance corresponding to the extent of the disease is often encountered in the right

iliac fossa. This area is generally oblong with the axis parallel to Poupart's ligament. The tenseness of the abdominal walls may interfere with the manifestation. If actual tumor is present it is generally palpable and is significant. Fluctuation is detected only in rare cases. Pain and tenderness are manifest on palpation. A difference in the temperature of the two sides is sometimes observable.

Percussion shows a localized area of impaired resonance surrounded by tympany. Marked flatness seldom obtains. Paralysis of the bowel with resultant distention increases markedly the tympany. Overlying intestine may mask the dullness.

Auscultation may aid percussion in delimiting the tumor. Distention due to accumulation of gas, and distention caused by intestinal paralysis may be differentiated by auscultation. In the first the peristaltic sounds are present; in the second, absent.

Differential Diagnosis. When the symptoms described are present the diagnosis is comparatively easy. When most of them are absent the cases require careful consideration. Sudden attacks of intestinal colic due to food irritation are most confusing. But the history of the case, the rapidly succeeding diarrhœa, the absence of tenderness in the right fossa and the relief which succeeds upon free purgation decide the matter.

Obstruction of the bowel may be mistaken for appendicitis, or *vice versa*. Obstruction is primarily afebrile, the pain may be located at any point, peristalsis is absent. Other features of that condition are detailed elsewhere. Fæcal accumulation should not be mistaken for tumor. Ovarian inflammation can be excluded by bimanual examination. Floating kidney is movable. In nephritic colic the pain is deeper-seated and radiates into the testicle and groin. The urine gives evidence of the kidney affection.

THE LARGE INTESTINE.

The colon begins with the cæcum in the right iliac fossa, passes vertically upwards on the right side to reach the under surface of the liver, traverses the cavity below the stomach to the spleen, makes a second rectangular bend and descends into the left iliac fossa as the sigmoid flexure and rectum. Its length varies extremely, being sometimes a trifle over a yard and at others being over two yards long. (1 to 2 m.)

The large intestine encircles the small intestine horseshoe-like.

Inspection. Occasionally in badly-nourished, marasmatic and rickety children the outline of the distended transverse colon may be distinctly seen. The peristaltic movements of the bowels may be seen occasionally in health and when increased are more visible.

Palpation. When the colon is distended with solid or semi-solid contents the enlargement may be felt. The fæcal contents are most apt to accumulate in the left iliac fossa, then in the right, but even the transverse colon may be filled. These accumulations are recognizable on palpation by their peculiar lack of elasticity, and although hard and resistent, when pressed upon by the ends of the fingers they indent like a hard snow-ball and the indentation remains after the removal of pressure (Simpson).

Percussion. The note of the empty colon is more truly tympanitic than that of the stomach; its quality is somewhat amphoric. By percussion the situation of the solid aggregations of the bowels may be marked out on the surface. Dullness is more apt to obtain over the descending colon than over the ascending, and Bennett points out the practical value of this fact in determining whether the patient should be given a purgative or an enema. If dullness is found in the right fossa an enema will fail to relieve the bowel and a purgative is indicated.

These accumulations might be mistaken for enlargements of the spleen and liver but their peculiar feel and their transitory nature should distinguish them.

Enteroptosis, or falling of the intestines, gives rise to no recognizable physical signs. Coloptosis, or descent of the large intestine, in which the transverse colon may lie upon the symphysis, may be recognized by percussion.

DYSENTERY.

Definition. An acute or chronic infectious inflammation of the large bowel characterized by frequent bloody, mucous or serous evacuations, associated with tormina and tenesmus.

Two forms are recognized, bacillary and amœbic. The disease is epidemic and universal. Shiga and Flexner have each isolated a bacillus.

Of the first form acute, catarrhal, croupous and hæmorrhagic varieties are recognized. Either form may be chronic. The liver is

often involved and abscesses, minute, multiple or large, of this organ are not infrequent.

Symptoms. Frequent stools, abdominal, colicky pains and fever mark the simple acute form. In the catarrhal variety the stools are more frequent, twenty to fifty daily, diarrhœal, consisting principally of mucus admixed with blood, and voided with much pain and tenesmus.

In the croupous form the stools are mucoid and bloody from the beginning and may soon consist almost entirely of blood. The evacuations are very frequent and accompanied by severe tormina and tenesmus. Recovery is slow. Death may occur from exhaustion; perforation, peritonitis or gangrene of the bowel may supervene.

Special Symptoms. Fever is slight in mild cases, moderate in severe cases, seldom exceeding 102°. Amœbic dysentery may be afebrile or the temperature may be comparatively high, reaching 104°.

In the simple catarrhal form pain is not severe; in the graver forms pain is excruciating, especially if due to ulceration, but is temporarily relieved by the passage of flatus or fæces. The passages are difficult and require much painful straining effort.

Tenderness is marked over the entire abdomen and with ulceration becomes exquisite. The patient lies on the back with the thighs flexed. Peristalsis is visible. Tenemus is almost continuous; paralysis of the bowel and prolapse of the rectum often occur. Nausea and vomiting complicate some acute cases, as do also strangury and vesical tenesmus. Necrosis, perforation and peritonitis are accidental complications not infrequent.

The stools at first are diarrhœal but fæcal matter may entirely disappear or be present only in minute quantities. They then consist of blood-stained mucus with perhaps small fæcal masses intermingled. Blood, either red or chocolate color, may be passed; tarry stools are not usual. In cases of gangrene necrotic masses with offensive odor, dark-brown or black in color, are voided. In the diphtheritic variety pseudomembrane and dead mucous membrane occur in the stools. Bacteria, amœbæ and other organisms abound.

Agglutination test. A specific serum reaction of the blood similar to the Widal reaction of typhoid fever is observed in most cases of dysentery, but in mild cases is slight or doubtful. It is limited to the bacillary disease and does not take place prior to the tenth day.

Differential Diagnosis. Cancer of the rectum is slower of devel-

opment, is accompanied by constipation, and digital examination or the proctoscope generally reveal the growth. Particles of the new growth are found in the stools and microscopic examination reveals their nature.

Cancer of the bowel gives no physical signs other than those of obstruction unless tumor develops. The discharges may contain fragments of the growth, mixed with pus and blood.

THE LIVER.

Perfect familiarity with the normal boundaries of this organ is essential to the study of the physical signs indicative of its maladies, since the diagnosis in cases of hepatic disease is chiefly determined by the recognition of alterations in its size, shape, consistency, position and the state of its surface, obtained by palpation and percussion. The percussion boundaries of the organ have been given in the section on medical anatomy. It is necessary to remember that the liver is suspended and very movable, altering its position under various circumstances. It is depressed with each inspiration, its lower border projecting below the ribs with a full breath. It recedes under the edge of the ribs on lying down, and when lying on the left side is not in close contact with the chest wall. In children the gland is proportionally larger than in adults and the edge is always palpable below the border of the ribs on the right, and the left lobe extends into the left hypochondrium.

While the upper boundary of the liver changes but little, except in conditions which cause depression of the diaphragm, as pleural effusion and emphysema, which have been discussed in the section upon lung and pleura, yet it is necessary to determine this outline. It is done by light percussion from above downward until the change of note in the overlying lung tells us that a solid organ has been reached. We percuss along the anterior median line, the right nipple-line, the mid-axillary line and the right mid-scapular line respectively, and draw a pencil through the points so determined. The boundary will be found generally to correspond to the base of the ensiform cartilage for the first point, the fifth right interspace for the second, the seventh rib for the third, and the ninth rib posteriorly for the last. The lower border is easily fixed by percussion, as the change from flatness to the tympany which marks the contact of the intestines with its free border is unmistakable. The normal width is about four

inches measured along both the nipple and the mid-scapular lines, and a trifle more in the axillary. In women who have been the victims of tight-lacing these widths are increased, but the constriction of the waist line will plainly indicate the reason. In extreme cases the right lobe is separated by a transverse furrow or band and the lower portion, which is abnormally movable may extend to the iliac border. The possibility of mistaking this artificial lobe for an abdominal tumor or movable kidney is corrected by following the free liver edge from the epigastrium downward and from the lumbar region forward until they unite at the doubtful point. In other cases a continuation of dullness on percussion without interruption to any considerable extent below the right border of the ribs points to the liver as the organ diseased.

Changes in the Position of the Liver. Elongation of the suspensory ligament causing floating liver is very rare. Displacement to the right or left or tilting of its lower edge is associated with various abdominal and thoracic affections.

Changes in the Size of the Liver. The liver is increased or diminished in size in almost every disease to which it is subject.

Its size is increased in the active congestion which accompanies many diseases, in passive hyperæmia, in all inflammations causing obstruction of the bile ducts, in abscess, in fatty and dropsical infiltration, in hypertrophy, in parenchymatous and amyloid degenerations, in hypertrophic and biliary cirrhosis, in cancer, cysts, and echinococcus cyst of the organ.

The volume of the liver is diminished in atrophy, in acute yellow atrophy and atrophic cirrhosis, in fatty degeneration and syphilis of the liver. Perihepatitis results in contraction of the organ by pressure, and tuberculosis of the liver is usually associated with cirrhotic contraction.

Passive hyperæmia. The liver is enlarged, tender and may be the seat of pulsations. The enlargement is subject to variations, ascites occurs late; jaundice is rare.

Obstruction of the bile ducts is associated with pain, fever and jaundice.

Abscess gives rise to pain or tenderness, fever, chill, sweats and jaundice. The enlargement may be upward into the axilla especially if the lateral or the posterior part of the right lobe is the seat of abscess; and the edge is pushed downward and appears below the

edge of the ribs. If the abscess is superficial and large, soft fluctuation is detected; in other cases tenderness and elasticity. The organ may reach enormous size.

Fatty infiltration produces a moderate, uniform enlargement which has a cushion-like feel. The normal shape is unaltered, the lower ribs do not flare. Ascites is not a sequence and the superficial veins are unaltered. The organ reaches the umbilicus and the edge is *rounded* and ill-defined; pain and tenderness are lacking.

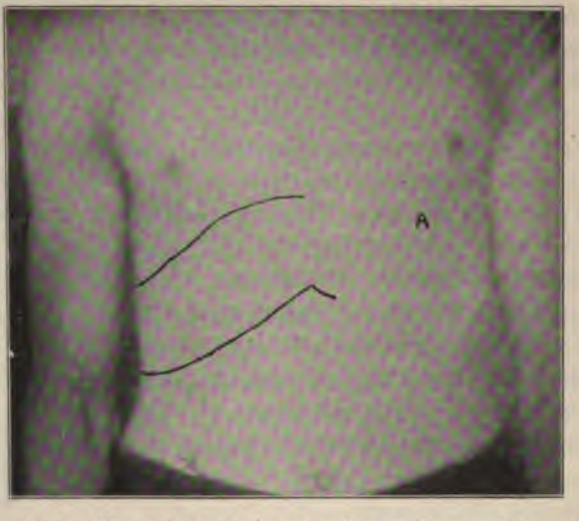

Fig. 68—The Liver—Normal area of percussion dullness. The apex impact is at A.

Dropsical infiltration accompanies general dropsy such as was described under dropsical effusions.

Amyloid liver reaches a size only surpassed by cancer of the organ. The enlargement is uniform in all directions and painless. It is of slow growth, extending over two or three years, and may fill the entire abdominal cavity. The tumor is characterized by its density and resistance. The edge is *sharply defined.* Ascites almost never occurs. The surface is perfectly smooth.

Hypertrophic cirrhosis occasions jaundice. The liver is enlarged but the surface is smooth. Ascites is absent.

Cancer gives rise to nodular enlargement of the liver, associated with tenderness, and enlargement of the superficial veins; jaundice

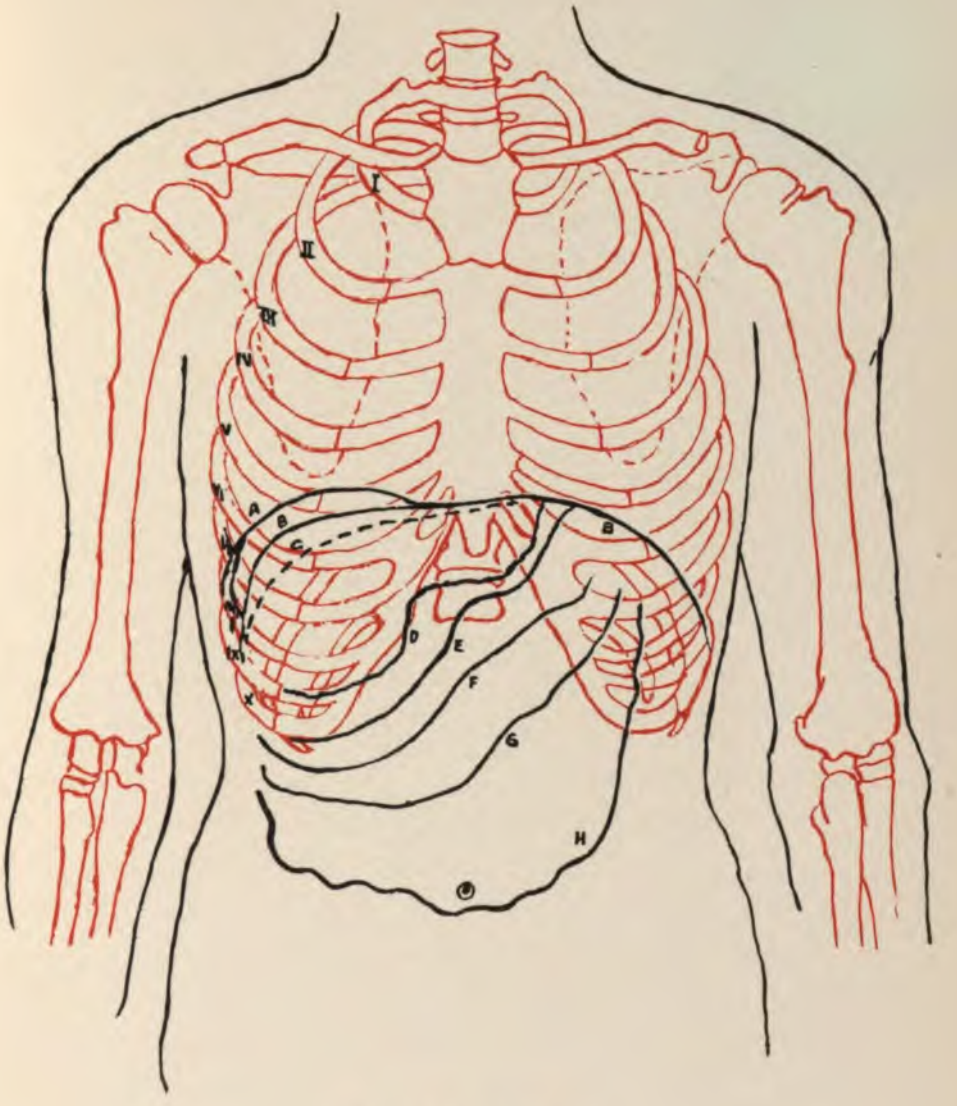

PLATE XXIII.

The Volume of the Liver in Various Diseases (after Rindfleisch).

A. The diaphragm displaced upward in carcinoma. B. Normal position of diaphragm. C. Line of relative dullness. D. Edge of liver in cirrhosis. E. Normal liver. F. Fatty liver. G. Amyloid liver. H. Cancer, leukæmia, and adenoma.

and ascites are both frequent. The liver enlarges into the left hypochondrium, pushes the diaphragm upwards and the free border descends even to the crest of the ilium. Cancer without nodular enlargements is recognized by the size of the organ, the cachectic symptoms and the jaundice.

Echinococcus causes enlargement with elasticity or fluctuation and the hydatid thrill may be felt by the superimposed hand on tapping over the enlargement. The thrill is a vibrating, tremulous movement similar to that obtained by tapping upon a bag of jelly.

Lymphadenomata and lymphomatous nodules occur in the liver in the course of leukæmic lymphadenomata, in typhoid fever and other infectious diseases.

Atrophic cirrhosis of the liver gives rise to hæmorrhages, abdominal dropsy which is profuse and quickly recurs after reduction, and œdema of the extremities. The caput Medusæ is developed. Jaundice is absent. The area of liver dullness may be determined by placing the patient upon the left side, so that the dropsical accumulation will gravitate away from the hepatic region. The intestines will float up and indicate by the characteristic tympanitic note the boundary of the liver. Usually the dullness stops about an inch above the lower thoracic border and extends no farther than the median line in front, instead of into the left hypochondrium.

After paracentesis the flaccid condition of the walls allows us to palpate the surface of the organ, which will be found to be rough and studded with small nodules.

Simple atrophy shows diminution in size of the organ, the surface remaining smooth, ascites being absent.

Acute yellow atrophy gives origin to few characteristic symptoms. The disease progresses with great rapidity, so that in a few days the area of liver dullness may be entirely wanting. The acute symptoms are those of a severe gastro-intestinal catarrh, jaundice, followed by abdominal pain, delirium, coma, hæmorrhage, from mucous membranes and even the skin. These, and the rapid course of the disease, are characteristic phenomena.

Syphilis may appear as atrophic cirrhosis resulting in irregular lobulated contraction more characteristic than the non-specific cirrhosis; as gummata, followed by cicatrices, or as diffuse connective tissue hyperplasia.

Differential Diagnosis. Conditions which may lead to the error that the liver is the seat of disease are:

(a) Displacements of the organ by diseases of the right side of the chest, as extensive pleuritic effusion and pneumo-thorax. Here we have the symptoms of these diseases to account fully for the change.

(b) Fæcal accumulations in the ascending and transverse colon; which are by no means easy to exclude as they give rise to a tumor below the edge of the ribs and the area of flatness and resistance seems to be continuous both to palpation and to percussion. The lack of elasticity and the indentations produced by palpation are aids. In case of doubt, enema and purgatives remove the source of error.

(c) Enlargement of the right kidney. When this organ increases considerably in size it may extend to the under surface of the right lobe of the liver. By placing the patient on his back and relaxing the abdominal walls the finger tips feel the apex of the kidney sloping downward and inward instead of outward and upward, and the fingers may pass vertically between the borders of the two organs.

(d) Cancer of the stomach is movable, its note is dullness overlying tympany and the two areas do not fuse.

(e) Enlargements of the spleen are told by the shape, the position and the increasing flatness which obtains as we approach the normal area of the organ.

THE SPLEEN.

Except in so far as it participates in the maladies originated by other organs, the spleen is subject to but few diseases, and owing to the comparative isolation of its situation, as well as what may be called its physiologic exclusiveness, these give rise to but few symptoms which serve to attract attention to the gland. For these reasons diagnosis of its morbid conditions often presents greater difficulties than is the case with any other abdominal organ.

Situation. This organ occupies the upper portion of the left hypochondrium. It is not palpable in its natural state and lies deeply imbedded between the fundus of the stomach and the diaphragm in the axillary line. Its outer, phrenic or convex surface rests against the convex arch of the diaphragm, which separates it from the thoracic wall below, while the upper portion of this same surface is separated therefrom by the further interposition of the pleura and to a slight

extent the edge of the lung. The external surface extends from the eighth to the eleventh rib. Its long axis is oblique and its direction and position nearly that of the tenth rib. In front lies the stomach and colon; to its inner side it is also in relation to the stomach as well as to the pancreas and left kidney. Vertically it lies between the ninth thoracic spine and the first lumbar spine. Its inner extremity is within two inches of the mid-dorsal line. Its outer extremity is

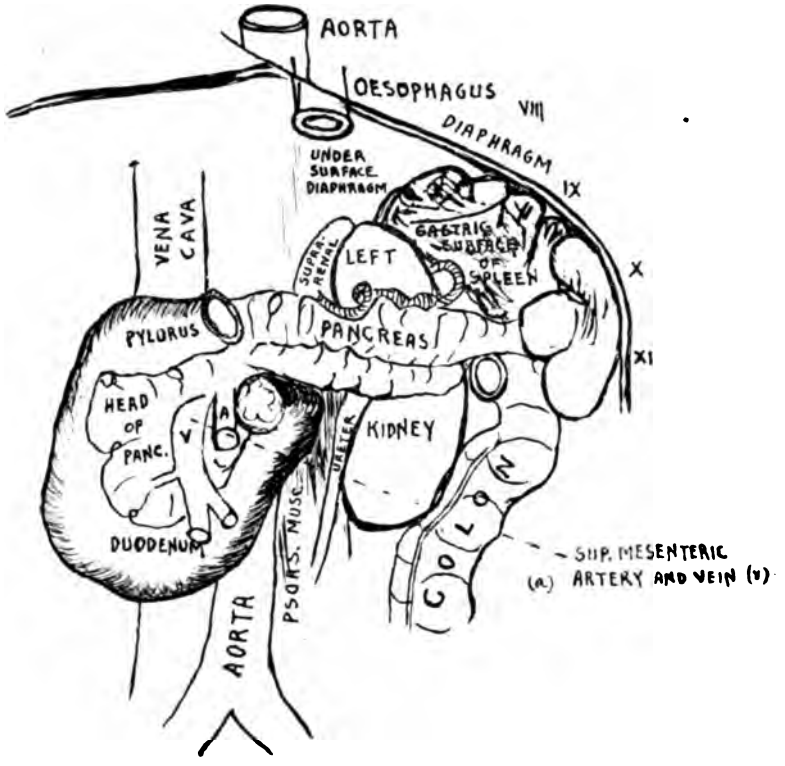

Fig. 69—The Relation of the Spleen to the left kidney. The figures refer to the ribs.

behind the mid-axillary line. A line drawn from left sterno-clavicular junction to the tip of the eleventh rib bounds it anteriorly.

Percussion area. To ascertain its boundaries place the patient on the right side. The anterior border is readily determined by the contrast between splenic dullness and the tympany of the stomach and intestines. Above, the limitation is determined by reaching pulmon-

ary resonance. Below, where the spleen and kidney are in contact it is impossible to separate the two by percussion. The area thus determined is about four inches long and three wide.

The spleen may be increased in size by disease, and atrophy is possible but would probably pass unrecognized in life. It is subject to acute swellings in the course of fevers and is chronically enlarged in the following conditions:

Congenital syphilis, and in acquired syphilis when amyloid degeneration attacks the organ; in alcoholic cirrhosis and in Hanot's hypertrophic cirrhosis; when it is the subject of tuberculous lesions as occasionally happens; in anthrax, as evinced by the older name for the malady—splenic fever. In these forms and in Hodgkin's disease and lymphatic leukæmia the spleen is moderately enlarged. In rickets it is often palpable. The organ is greatly enlarged in spleno-medullary leukæmia and in palludism, in which its size takes second rank. In typhoid fever it is considerably enlarged at the end of the first week and is palpable unless pushed back under the diaphragm by a distended colon, in which case the normal area of dullness may be lessened or even disappear. In death after the fourth week, atrophy of the spleen is not rare.

Enlargements of the spleen are smooth, uniform and solid, without either pain or tenderness. The oblong mass is felt directly under the integument extending from under the ribs on the left side forward towards the median line, which it may reach, or downward towards the crest of the ilium, and can scarcely be mistaken or overlooked.

The upper edge is rounded, the lower sharp and the notch or fissure may be apparent. The tumor is somewhat movable.

The tumors which might be mistaken for enlargements of the spleen are enlargement of the left lobe of the liver, fæcal accumulations, ovarian disease, enlargement of the left kidney and possibly cancer of the cardiac end of the stomach. The first can be distinguished by tracing the organ across the median line to the right hypochrondrium where it connects with the right lobe. The differential points of the second have been named. The character of ovarian tumors will be considered hereafter. The left kidney may enlarge forward towards the left hypochrondrium and assume a position analogous to that of the spleen, but the tumor may be traced backward into the loin, where will be found its chief bulk, and is much more fixed.

Tumors of the spleen enlarge forward and whenever the enlargement is considerable, project from under the ribs and fall forward when the patient is placed in the knee-breast position. Cancer of the stomach could not readily be mistaken for splenic enlargement unless attachments had formed, which is unlikely. The dull note overlying large tympany on deep percussion aids in the decision.

THE KIDNEY.

The situation of the kidneys has been given with sufficient accuracy in the section upon medical anatomy. The right is in relation above with the liver, below with the cæcum, anteriorly with the ascending colon, behind with the diaphragm. The left is in relation above with the spleen, in front and below with the colon, behind with the diaphragm.

It is by no means a simple matter to determine by percussion the normal outlines of the kidneys in well-developed muscular or corpulent individuals, much less to ascertain diminution of their volume. By palpation and percussion the normal areas are obtainable, and in spare subjects and persons with well-relaxed abdominal walls they may be ascertained with considerable accuracy. In enlargement of the organs the task is sometimes easier.

Inspection rarely furnishes any evidence.

Palpation from behind is often negative; in enlargements the external borders may be felt to project beyond the free edge of the quadratus lumborum. When, however, the patient is supine, one hand being placed in the lumbar region, with the other pressed firmly downward and inward the organ is felt, and if enlarged the tumor may be pressed backwards and by bi-manual palpation its size, situation, consistency and possibly its nature determined.

Percussion. The position of the patient should be prone, which allows the intestines to float upward, the spleen and liver to project forward, and any accumulation of fluid which may be present to gravitate away. The external margins are indicated by a tympanitic note. Inwardly the dullness coalesces with that of the vertebræ.

Enlargements will increase the lateral area of dullness.

Absence of one kidney is frequently observed. The opposite organ may be hypertrophied.

Displacement of the kidney is congenital or acquired. The dis-

placement may be in any direction; most frequently it is into the pelvis.

Acquired malpositions are oftenest met with in women after repeated pregnancies, or caused by tight-lacing, diseases and displacements of the liver. The right is more frequently displaced than the left.

When the elongation of the peritoneal reflection is such that the kidney descends into the abdominal cavity, it may be readily felt through the anterior abdominal wall by placing the patient on the back with the thighs flexed, grasping the tumor in the palm of the hand. The normally dull area in the lumbar region is replaced by tympany. The organ's size, shape and smoothness distinguish it from mesenteric tumors. When pressed upon it glides away into the abdominal cavity and wholly disappears.

The kidneys are enlarged in active and passive hyperæmia, in œdema, in acute diffuse and acute catarrhal nephritis, but in none of these is the increment possible of detection by the physical signs. In hypertrophic and amyloid kidney, in which disorders the organ may be enlarged to twice its normal bulk, the enlargement should be detected. Sarcoma, carcinoma, calculus pyelitis, hydronephrosis, cystic dilatation due to obstruction, or congenital cysts, the latter affecting both kidneys, are of large size and the tumor readily diagnosed. Echinococcus cysts are rare.

THE BLADDER.

When empty the bladder reposes behind the symphysis and cannot be detected. When distended it rises above the pubes and a visible tumor appears in the hypogastrium, which is elastic, smooth and moderately hard on palpation. On percussion its dome-like area is easily determined by its flat note contrasted with the tympany of the intestines in contact with it. In children the bladder is normally higher than in adults and can be detected even when partially filled. In females an ovarian tumor or an enlarged uterus might be mistaken for a distended bladder, but the catheter would resolve the doubt.

THE UTERUS.

The normal, unimpregnated uterus is situated deep in the hypogastrium and is inaccessible to external palpation or percussion.

When enlarged from any cause its boundaries are determined by

these methods with readiness and exactness; and when impregnated auscultation furnishes us with confirmatory evidence. Pregnancy cannot be differentiated from other smooth, uniform, progressive enlargements of the uterus by palpation or percussion, yet the shape of the enlargement is nearly if not quite distinctive. The pregnant uterus is egg-shaped with the large end up, while the swelling of ascites, which gives to the abdomen a similar appearance when the patient is erect, with the patient on the back, is flat, the flanks flaring. Tumors of the womb are seldom symmetrical and regular. Ovarian tumors are spherical.

After the fourth month of pregnancy is past adventitious sounds are to be heard on auscultation of the uterus containing a living fœtus, viz: The placental bruit and the fœtal heart sound. The patient is placed upon the back with the thighs flexed, the abdomen uncovered. With the stethoscope different areas are carefully and attentively examined until the presence or absence of the sound is determined. The areas over which they are heard are circumscribed and their intensity is not great, hence care must be exercised lest they be overlooked.

The placental bruit may be heard at an earlier period than the fœtal pulse. It is an intermittent, blowing sound, single, and was called the *bruit de souffle*, or bellows murmur, by its discoverer, M. Kergaradec (1823). It is sometimes hissing. In time it is synchronous with the maternal pulse. It varies in character and is sometimes present and disappears at others. Also its seat and the areas over which it is heard vary in different cases. Delens asserts that it can be heard as early as the third month. During labor its intensity is greatly increased. A similar bruit is sometimes present in uterine and ovarian tumors.

The fœtal heart sounds have long been likened to the ticking of a watch enveloped in a napkin. They may be heard in the course of the fourth or fifth month (Cazeaux). The pulsations are double, short, and rapid and vary from 130 to 160 per minute. The two sounds vary in intensity, the second being the clearer, more sonorous and more distant. They are most often audible on the anterior, inferior abdominal wall, just above the iliac fossa, but the location varies. Twin pregnancies may occasionally be foretold by hearing the heart sounds at distant points over the tumor and by the absence of synchronism in the two sounds. The pulsations of the maternal arteries

could be mistaken for the intra-uterine sounds only when the pulse is abnormally rapid, and even then the quality would materially differ. When inaudible in one position the fœtal sound may be heard by changing the position of the mother. Total cessation after having been present, indicates the death of the fœtus.

Tumors of the Uterus. These occupy the same area as the uterus enlarged by pregnancy. Their outline can be established by palpation and percussion. The peculiar hardness and elasticity, the position and shape, readily establish their connection with the womb. Doubt is dispelled by external palpation and the use of the uterine sound. Irregular and nodular growths can be felt through the abdominal wall.

Ovarian Tumors. The ovaries, like the bladder and the uterus, in the normal state give no evidence of their existence to external palpation. When, however, they become enlarged by disease, they rise from the pelvis into the abdominal cavity and their outline becomes apparent on palpation. Percussion gives a sharp line of demarcation, owing to the growth being surrounded by intestines. When ovarian growths reach enormous size they occupy the entire anterior abdominal cavity, pushing the intestines backward and upward into the dome of the diaphragm and raising that structure to its highest possible stretching point. When small and first attract the attention of the patient they may be confined to one or the other iliac region, but oftenest they are central when first observed. They are of frequent occurrence, and as the medical man is most often first sought for advice, a few salient points upon their diagnosis is not amiss.

If the tumor has not risen from the pelvis a bi-manual examination will reveal that the growth is lateral and not central; and if the former, the notch or interval which separates it from the uterus is felt. By palpation and the aid of the sound the independent mobility of the two is established. The sound shows increase in the depth of the uterine cavity in tumors of that organ, which is absent in ovarian disease.

When the tumor has risen into the abdominal cavity inspection in the earlier stages shows the uneven and unsymmetrical development of the tumor due to its occupying the iliac or lumbar region of one side, whence it grows towards the umbilicus. Later, when the entire abdomen is occupied, the tumor projects as a smooth, spherical, fairly symmetrical swelling which differs from the central rounding of pregnancy and from ascites in the manner stated.

Palpation. Small ovarian growths are firm and elastic, the large ones fluctuating and soft. When only a portion of the abdomen is occupied by the tumor its outline can be determined by palpation and percussion. The sense of fluctuation varies, increasing with the size, but is usually quite distinct in all cases.

Percussion gives a flat sound over the area occupied by the tumor, with tympanitic resonance over the areas occupied by the intestines.

Diagnosis. The differentiation of ovarian dropsy from ascites is easy, yet confusion frequently arises. Hence the following distinctive points are noteworthy.

Inspection. Ovarian tumors project forward and are rounded and central whatever be the position of the patient. In ascites the form changes with the position: when the patient lies on the back the enlargement is uniform and the abdomen is flattened.

Percussion made in the supine position gives dullness over the surface of an ovarian tumor, while in ascites, because the intestines float on the fluid, the resonance is tympanitic *above* and *dull* as high as the fluid extends.

Change of posture alters the area but little in ovarian dropsy but markedly alters it in general dropsy.

These points and the evidence of cardiac, renal or hepatic disease to account for the ascites should enable one to decide the question. Renal cysts push the intestines forward, not backward; and pus, blood and albumin appear in the urine. They grow slowly from above downward, and when pushed up resonance appears between the tumor and the pelvis. The same points apply to splenic and hepatic cysts. To mistake pregnancy for ovarian cyst would be inexcusable carelessness. It should be borne in mind that either may arise in the case of unmarried females.

For additional points upon the diagnosis of ovarian cysts from cysts of the broad ligament and from uterine fibro-cysts, the reader is referred to works on gynæcology.

The omentum may be the seat of tuberculous disease and deposit, of sarcoma, cancer and of hydatid cysts. The thickening and nodular enlargements are easily felt through the abdominal walls in cancer and may often be seen. They give rise to a dull area on percussion in the upper zone of the abdomen which increases with their growth from above *downward,* and unless extensively adherent they may be pushed upwards. The growths are superficial and generally tender.

Cancer and *tuberculosis* of the omentum give rise to ascites and the general symptoms of the diseases. In a case of cancer occurring in my own practice, small, hard nodules were early detected in the omentum, which gave the sensation on palpation of small chips of wood laid upon the intestines.

Percussion in cancer is woodlike, overlying tympany, until the fluid accumulates.

ASCITES.

An accumulation of serous fluid free within the abdominal cavity is termed *ascites.*

The causes are local and general.

Local causes are: Inflammations of the peritoneum, simple or tubercular; cancer of the peritoneum.

Obstruction of the portal circulation within the liver, as in cirrhosis and chronic passive congestion, or, in the branches of the gastro-hepatic omentum by new-growths, peritonitis or aneurysm.

Pressure upon other vessels, as in the ascites associated with ovarian dropsy, due to pressure exerted by the growth.

Ascites accompanying enlargements of the spleen.

General causes are those which produce general anasarca, and include organic heart and lung diseases, chronic Bright's disease, and hydræmia. Here the cause may operate directly or mechanically. In heart disease, when ascites occurs without general œdema, secondary changes in the liver should be sought for. Pulmonary emphysema and cirrhosis of the lung exhibit ascites as terminal symptoms. The various kidney lesions associated with ascites have been mentioned.

Inspection. The abdomen is uniformly enlarged, and when the patient lies on the back the flanks flare outward. The umbilicus is prominent and protrudes on standing. In old and recurrent cases the lineæ albicantes show. In cirrhosis, the superficial veins are prominent and the caput Medusæ may be present (see Liver Diseases).

Palpation. Fluctuation is easily felt by placing one hand over the lateral area and smartly tapping on the opposite side. The fluctuation is more apparent if the patient sharply depresses the linea alba with a thin book or cardboard.

Small effusions are palpable by placing the patient in the knee-breast position when the fluid gravitates to the dependent part and floats the intestines away. In case of large effusions difficulty may

be experienced in palpating the abdominal organs. This may be overcome by suddenly deeply indenting the walls with the finger-tips in the desired locality, in order to displace the fluid. In cases of considerable tension even this fails.

Percussion. The area of dullness surrounds the tympanitic area of floating intestines when the patient is in the dorsal position, hence the lateral areas are dull, and the height of the dullness rises as the fluid increases. The line separating dullness and tympany is sharply-defined, and when marked out is ovoid in shape.

The dullness is movable. When the patient is placed on the side the region of the upper flank, previously dull, becomes tympanitic. Effusions too small to give rise to dullness with the patient on the back are easily discovered by placing him in the knee-breast position.

The diagnosis of ovarian ascites from free peritoneal ascites has been given. The principal diagnostic point is the recognition of the causative condition. The heart and lungs show positive signs of disease when the dropsical stage is reached, and the dyspnœa is out of proportion to the pressure caused by the fluid. The liver shows enlargement and the superficial veins are its sign-board. The urine gives evidence of Bright's disease. Cancer of the omentum can be felt as lumpy enlargements directly under the skin. Fever, hectic, wasting and night-sweats point to tuberculosis. Percussion shows enlarged spleen. Blood examination demonstrates its relationship to the condition.

SECTION XIV.

EXAMINATION OF THE STOMACH CONTENTS.

Introduction. The gastric fluid is a thin, almost colorless liquid, acid in reaction, with a sp. gr. of 1001 to 1010. The analysis varies greatly with different conditions and different observers. The following is approximate (Schmidt):

Water	994.4
Organic substances, pepsin	3.2
Hydrochloric acid	0.2
Sodium chloride	1.5
Potassium chloride	0.5
Other inorganic salts	0.2
	1000.

The total acidity varies between 0.10 and 0.30. The free acid varies between 0.10 and 0.30, normally only reaching the latter point on a carbo-hydrate diet. Ordinarily on nitrogenous diet it does not exceed the minimal figure.

The secretion of gastric juice begins immediately upon the ingestion of food but the hydrochloric acid contained in the first portions secreted combines rapidly with the ingested proteids and their contained inorganic salts, the product being acid proteids and acid salts. The chief mineral salts are the di-sodic and di-potassic phosphates, which are converted into the corresponding acid phosphates. For this reason free hydrochloric acid is not found in the stomach contents until a varying time after eating, depending upon the character and amount of food ingested. After an ordinary meal, free acid appears in healthy stomachs in from forty-five minutes to one hour, and the amount gradually increases as digestion proceeds, reaching its height towards the conclusion of gastric conversion.

Fats and starches in superabundance delay its appearance. After the Ewald test-meal free acid should appear in twenty minutes.

With ordinary diet lactic acid is very generally found in the stomach contents in small amounts, and under certain circumstances butyric and other organic acids may appear, without being manifestations of disease. In gastric catarrh, anæmia, during the course of fevers, pepsin and hydrochloric acid are considerably reduced. Hydrochloric acid is increased in gastric ulcer and in nervous dyspepsia (hyperchlorhydria), and in those queer cases of super-secretion of Reichmann and Rossbach (gastroxynsis).

Fermentative changes may cause lactic and butyric acid to appear in large amounts in the fluid, accompanied by gaseous distention and sour, gaseous eructations. Excessive mucus accompanies catarrhal inflammations. Blood, bile, fragrants of growths, albumin, ammonium carbonate and urea occur in special conditions.

The clinical examination of the gastric fluid is practically limited to the determination of:

(a) The reaction.

(b) Total acidity.

(c) Presence of free acid and of acid salts.

(d) Presence of free hydrochloric acid.

(e) Organic acids.

(f) Presence of syntonin, peptone and the pepsin strength.

The volumetric analysis can be performed readily by those possessing even slight skill in laboratory manipulations. The necessary apparatus is simple and inexpensive. The standard or volumetric solutions are easily prepared or may be purchased from competent pharmacists.

The necessary apparatus is:

A 50 c. c. burette, graduated to 0.1 c. c.

A graduated pipette, and one or two graduated cylinders; one or two flasks, capacity 500 c.c.; or a litre, and necessary test tubes.

EXAMINATION OF THE GASTRIC CONTENTS.

The examination of the gastric contents for clinical purposes, such as ascertaining the state of the secretion, the motor function of the stomach, the presence or absence of abnormal substances and the products of gastric digestion, has approached almost to a routine procedure. After fasting all night, or, better, after washing out the

stomach, the patient is given a test-breakfast. Ewald's is simplest and most used, consisting of an ordinary baker's roll eaten dry, and 300 c.c. of weak tea, without milk or sugar, or a like quantity of warm water. Such a "meal" contains little or no lactic acid, the presence of which is the objection to most proposed test-meals. The Boas' test-meal, consisting of a gruel made of a tablespoonful of oatmeal boiled in a litre of water, may be used for estimating lactic acid. One hour after ingestion the residue is removed with the stomach tube and examined. The residue should be about 40 c. c. After noting the quantity, odor, color, consistence, and amount of mucus, a small portion is examined with the microscope to determine the nature of the

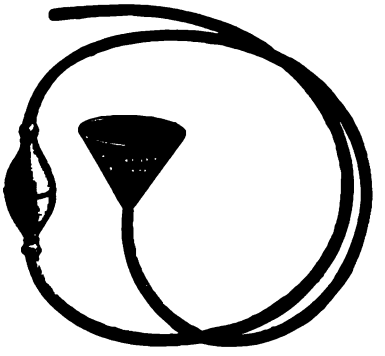

Fig. 70—Stomach Tube with Syphon Bulb.

residue and the presence of anatomic elements. The remainder is well-shaken, filtered, and the clear filtrate tested as soon as possible.

Reaction. The normal reaction of the gastric juice is decidedly acid, due to hydrochloric acid, acid salts (NaH_2PO_4), occasionally to organic acids and carbonic acid. During digestion free organic acids, acid albumins, consisting of combinations of hydrochloric acid and organic acids with the proteids of the food, and the acid salts (acid sodium and acid potassium phosphates) also add to the acidity. Lactic acid should not be present after the oatmeal breakfast. Its sources are, fermentation of the stomach contents due to retention—deficiency of the motor function and to pyloric obstruction—and to the introduction of sarcolactic acid into the stomach with foods. Butyric and acetic acid are fermentation products. In gastric carcinoma, lactic acid is found after the test-breakfast. The reaction is

determined with litmus paper which acids turn red. Other substances used as acid indicators are:

Name of Dye	Solvent	Color with Acids	Color with Alkalies	Reacts with	Sensitiveness
Phenolphthalein	1% in Alcohol	Colorless	Pink or Red	All Acids	Extreme
Congo-red	Water 1% Sol.	Blue	Claret	Free Acids Only	H Cl 0.1 in 1000 Lactic 0.2 in 1000
Tropeolin	Water	Red	Yellow	All Acids	H Cl 0.3 in 1000
Litmus	Water	Red	Blue	All Acids	Extreme
Dimethyl-amido-azobenzol	0.5% in Alcohol	Cherry	Yellow	Free Mineral Acids	H Cl 0.2 in 1000
Alizarin-sulphonate of sodium	Water 1% Sol.	Yellow	Violet	Free Acids and Acid Salts	H Cl 0.2 in 1000
Boas' resorcin sol., Resorin, 5 gm.; cane sugar, 3 gm.; alcohol, (95%), 100 c. c.	Alcohol	Pink	Colorless	Free Mineral Acids	H C l 0.5 in 1000
Gunzburg's Solution. Phloroglucin, 2 gm.; vanillin, 1 gm.; Alcohol (95%), 100 c. c.	Alcohol	Pink	Light Brown	Free Mineral Acids	H C l 0.5 in 1000

Dried papers colored with the above solutions are convenient for testing. By testing the filtered gastric contents successively with three papers colored with the above dyes, we may determine whether it is acid (litmus); whether the acidity is from free acid (congo-red); and whether free hydrochloric acid is present (azobenzol). If the congo-red paper, blued by free acid, is gently warmed the blue color is discharged when it is due solely to organic acids, but remains if produced by hydrochloric acid. Boas' resorcin solution is more stable than Gunzburg's solution, and equally sensitive. It is used thus:

A few drops of the reagent are spread upon a porcelain dish, which is gently heated; a glass rod dipped in the gastric fluid is drawn across the field. Free hydrochloric acid causes a scarlet streak to appear. If the acid is present in small quantity the streak appears only on complete evaporation. Previous filtration is not necessary. Proteids, acid salts and organic acids do not interfere with the test. One part of hydrochloric acid in 20,000 parts of water is detected by the method.

Organic Acids.—The sources of lactic, acetic and butyric acid have been mentioned. The presence of more than a trace of organic

acid in the stomach after the test-meal should be regarded as pathologic. Butyric acid has an odor like that of rancid butter and can generally be detected by the smell. Acetic acid has a vinegar-like odor, which is emphasized by boiling.

The tests may be applied to the mixed fluid or the organic acids may be extracted with ether.

Method: 5 c. c. of the gastric fluid are boiled with three drops of hydrochloric acid until syrupy in consistence. After cooling add a small quantity of ether, shake well, decant into a basin, add ether again and repeat. The ether is evaporated, the residue redissolved in a few drops of water and tested.

The most important of the organic acids is lactic acid and it occurs in larger amount and more frequently than the others. As stated, the presence of organic acids results whenever the food is too long retained in the stomach, and occurs when hydrocholoric acid is deficient, in dilation of the stomach, deficiency of its motor function, in cancer and in stenosis of the pylorus.

The breakfast of oatmeal-gruel contains no lactates, and is superior to the Ewald meal.

Tests: Dilute solutions of ferric chloride turn canary-yellow on contact with lactic acid (Uffelmann's test). A few drops of dilute neutral solution of ferric chloride are mixed with a fourth the quantity of carbolic acid, and water added until a clear amethyst color is obtained. Lactic acid in the proportion of 1 to 2,000 instantly changes the blue color to yellow. Lactates produce the same result, but as their clinical significance is the same, it does not interfere with the test. Sugar, alcohol and certain salts, as phosphates, are capable of changing the color. Butyric acid, 5 parts in 1,000, changes Uffelmann's reagent to a tawny-red color. The quantitative determination of organic acids is rarely necessary for clinical purposes.

(a) *Quantitative estimation of the total acidity.*

Method: 10 c. c. of the filtered fluid are accurately measured into a beaker and three drops of the phenolphthalein solution are added. The burette is filled with $\frac{N}{10}$ NaOH solution* which is added until

*Which means a decinormal solution. A normal solution is made by dissolving the molecular weight in grams of this substance in a litre of water. A decinormal solution is one-tenth of that strength. In the case of NaOH, 40 grams to the litre constitutes a normal solution, but owing to the impossibility of obtaining pure sodium hydroxide, the solution must be standardized by titration against a normal solution of oxalic acid and corrected as follows: 50 grams.

a permanent pink color is produced in the beaker. Near the completion of the test, each drop produces a pink cloud which disappears on stirring with the glass rod, until neutralization occurs, when a single drop produces a permanent shade. The number of c. c. of the decinormal alkaline solution used multiplied by .00364 equals the weight in grams of hydrochloric acid in ten c. c. of gastric fluid. This multiplied by ten gives the percentage.

The decimal .00364 represents the weight of HCl. neutralized by 1 c. c. of the NaOH. solution.

(b) *The estimation of free hydrochloric acid.*

Töpfers method: To 10 c. c. of gastric fluid add three or four drops of a 0.5 per cent. alcoholic solution of dimethyl-amido-azobenzol. The decinormal solution of NaOH is run from the burette until the red color changes to a pure yellow. This reaction is not affected by combined hydrochloric acid nor organic acids, unless 0.2 per cent. of lactic acid be present. The calculation is made by multiplication as before.

The test may be made by using Boas' resorcin solution if preferred. A few drops of this solution are spread on a piece of ground glass and dried. The $\frac{N}{10}$ NaOH solution is added from the burette to the 10 c. c. of filtered gastric fluid minus the indicator, as before, until a drop of the solution removed with the rod fails to react by giving a pink color when drawn across the warmed plate. Blowing upon the plate intensifies and hastens the color production.

(c) *Estimation of combined hydrochloric acid.* Here alizarin-sulphonate of sodium in 1 per cent. aqueous solution is used as an indicator, which reacts with all acid elements except acid albuminates.

The alkali is added as before to 10 c. c. of gastric fluid, colored with three drops of the indicator, until a clear reddish-violet color is attained, which is not reached *until free acid, acid salts and organic acids* have been neutralized. As the color produced in the alizarin test is not easily recognized by the inexperienced, a 1 per cent. solu-

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of best NaOH are dissolved in a litre of water. 10c.c. of this solution are placed in a beaker and 3 drops of the 1 per cent. alcoholic solution of phenolphthalein are added for an indicator. From the burette is dropped sufficient of the normal oxalic acid solution (63 grams to 1 litre) to just discharge the pink color. If each solution were *normal* equal volumes would exactly neutralize each other. Suppose 12c.c. of the acid were required, then the NaOH solution is 12-10 strength instead of 10-10, hence must be weakened by additions of the difference in c.c. of water, which is in this case 2-10. In other words 20 c.c. of water must be added to every 100c.c. of the hydroxide solution to render it normal.

tion of sodium phosphate or sodium carbonate may be colored with the indicator and used for comparison. It will be seen that the difference between the number of c. c. used in this titration and that used in the estimation of total acidity by the (a) phenolphthalein method equals the number of c. c. necessary to neutralize the combined hydrochloric acid. Hence by these three tests we get the total acidity (a), the free acid (b), the combined acidity by subtracting (c) from (a). The organic acids and acid salts together are found by adding (b) to (a) minus (c) and subtracting the sum from (a).

Free acids and acid salts. A simple method of determining whether acidity is due to free acid or to acid salts is by the addition of calcium carbonate, $CaCO_3$. Free hydrochloric acid decomposes the carbonate and is neutralized, while acid sodium phosphate, NaH_2PO_4, is unaffected. If therefore the acidity disappears it was due to hydrochloric acid, if diminished, as shown by the color reaction to test-paper, the remaining acidity is due to acid salts. The test may also be used quantitatively by determining the total acidity before and after the addition of the carbonate (Leo's test).

Significance of hydrochloric acid. In the acute stages of fevers, hydrochloric acid is temporarily diminished. In dilatation of the stomach, atrophic gastritis, amyloid degeneration, some forms of nervous dyspepsia, anæmias, cachexias, and Addison's disease hydrochloric acid is permanently diminished. It is generally absent in cancer, especially if accompanied by extensive gastritis, and may be absent in chronic gastric catarrh and extensive degeneration.

DIGESTION PRODUCTS.

To determine the efficacy of the digestive function we may examine the products formed in the stomach during the conversion of proteids into peptones. When the albuminous compounds named proteids are submitted to the digestive action of pepsin, trypsin, rennin and certain other enzymes, intermediate products are produced in the course of the conversion, which finally become peptones. These intermediates are termed albumoses or proteoses and the process is named proteolysis. When pepsin in the presence of hydrochloric acid is the proteolytic agent a small proportion of the proteid is first converted into acid-albumin, which may be precipitated by neutralizing the fluid during the early stages of digestion. At a later stage it disappears and other albumoses appear as the result of hydrolysis.

Some of these substances should be present in the residue of the test-meal removed at the end of an hour, others do not appear until a much longer period. Native proteids, albumin and globulin are precipitated by slightly acidulating and boiling.

Acid albumin is precipitated by exactly neutralizing the solution. The $\frac{N}{10}$ NaOH may be used with phenolphthalein as the indicator.

Primary albumoses, which may be taken to represent the next step, are obtained by first removing the native proteids and acid albumin as above, then saturating the filtrate with $MgSO_4$.

Secondary albumoses. Filter out the above primary albumoses and saturate the filtrate with $(NH_4)_2\ SO_4$. Filter and test the filtrate for peptone. Peptones are tested for by the biuret reaction, viz: To the above filtrate add one c. c. of KOH or NaOH and two drops of $CuSO_4$ solution, a rose or red color indicates peptone.

Little peptone is found in the stomach at any time as the result of gastric proteolysis. Whether this is due to the fact that the process stops short of its formation, as is held by Ewald and Gumlich, or to the immediate absorption when formed, is undecided. Even from the small intestine it rapidly disappears.

Starch. The salivary diastase continues the conversion of starch into dextrin and maltose until free hydrochloric acid appears, which after an ordinary meal may be delayed forty minutes to one hour, when the conversion should be complete. The process is called amylolysis. At the end of this time iodine should give no blue color. Erythrodextrin gives a reddish-violet color with aqueous solution of iodine. The blue or purple reaction indicates faulty or deficient diastase in the saliva or excessive acidity of the gastric juice.

Pepsin. The efficacy of the pepsin is tested by noting its action on discs of coagulated albumin—the white of boiled eggs. When hydrochloric acid is present pepsin is rarely absent from the gastric juice. In its absence pepsinogen may be present and is converted into pepsin by the hydrochloric acid. Two tests should be made; one with 5 c. c. of the filtered fluid, the other with the fluid plus two drops of hydrochloric acid, using discs of equal size and thickness, cut from the egg. The test tubes are kept at a temperature of 100 F., and examined occasionally to ascertain the effects. If no liquefaction occurs, pepsin is absent. If liquefaction occurs only in the tube to which acid has been added, this substance is needed to develop the ferment.

Rennin. Carefully neutralize 5 c. c. of gastric fluid and also 5 c. c. of milk. Mix: if rennin is present coagulation occurs in ten to fifteen minutes.

The rapidity with which stomachic absorption takes place is ascertained by noting the time at which the iodine reaction occurs in the saliva after administration of potassium iodide. A capsule, carefully wiped, containing three grains of KI is administered and at the end of five minutes and each succeeding minute, the saliva is tested with strips of bibulous paper which have been previously soaked in starch paste and dried. The papers are moistened on the tongue, then touched with a drop of commercial nitric acid, which always contains traces of nitrous acid. Iodine is liberated and a blue spot is produced. The reaction should occur in ten to fifteen minutes. When the reaction is delayed twenty minutes or more absorption is abnormally delayed and indicates disease of the mucosa.

The motor function of the stomach is conveniently determined by Ewald's salol test or by Fleischer's method.

Gastric peristalsis pumps the portions of prepared food products which are not directly absorbed from the stomach, through the pylorus. The stomach peristalsis begins about fifteen minutes after food ingestion, and gradually becomes more and more energetic until the end of stomach digestion, which lasts four or five hours. Dilatation of the stomach, weakening or degeneration of its muscular coats and pyloric stenosis interfere with the food transfer, and the contents are retained abnormally long in the stomach. On the other hand I have frequently found that in persons suffering from intestinal indigestion of the catarrhal order, with the production of diarrhœa, the stomach peristalsis is abnormally active. Fleischer administers a capsule containing about two grains of iodoform, which is not decomposed in the stomach but which is converted into iodide of sodium in the duodenum. The capsule is administered with the test-breakfast and iodine should appear in the saliva in from 55 to 100 minutes.

Both the motor and absorption powers are determined by removing the residue of the test-breakfast after the lapse of stated times, and noting the amount. After two and a half hours, there should be no residue.

Klemperer introduces 100 c. c. olive oil into the washed-out

stomach and removes the unabsorbed portion after two hours, to determine the amount.

Ewald administers a capsule containing ten grains of salol, which is not absorbed in the stomach, but which splits into phenol and salicylic acid in the duodenum. Salicylic acid should appear in the urine in one hour or one hour and a quarter after administration. A drop of urine is caught on a piece of filter paper. On touching the middle of the drop with a drop of 10 per cent. solution of ferric chloride a violet rim appears around the spot.

Mucus. When not apparent to the eye in the vomit or in the residue, mucus is not in excess. It is increased in all catarrhal and inflammatory conditions and then occurs in ropy, stringy masses. In chronic alcoholics and drug habitués it reaches excessive proportions. Mucus is recognized by shaking the sediment with dilute NaOH, filtering and precipitating the dissolved mucus by the addition of acetic acid.

MACROSOPIC EXAMINATION OF THE RESIDUE.

The quantity, character, amount, color, odor and proportion of mucus are noted. The residue removed five or six hours after an ordinary meal furnishes us more information upon these points than does the test-meal. Particles of food from preceding meals may be found. In cases of acute summer gastritis it is not rare to find in the vomit remnants of food ingested twenty-four to forty-eight hours previously. Excess of proteids indicates deficient acidity. Excess of starch indicates excess of acidity.

Bile and blood are visible to the eye when present in any quantity.

MICROSCOPIC EXAMINATION.

Food particles may be identified, blood and pus cells, bacteria, yeast-cells, sarcinæ, bile stains and sometimes particles of new-growths may be recognized. Staining with methyl-blue best demonstrates bacteria, which are always present in the stomach and are undoubtedly necessary to the digestive function. Only when in excess or when specific forms, the product of specific disease, are present can they be considered pathologic. The Boas-Oppler bacillus is found in carcinoma, and its absence is generally said to indicate the absence of the growth, although Osler lays more stress upon the presence of lactic acid, which the bacillus is thought by its discoverer to

be the chief instrument in producing, than upon the bacillus itself. Sarcinæ are in excess in pyloric obstruction and dilatation. Fermentation is due to bacterial activity and produces the well-known train of symptoms named dyspepsia.

SECTION XV.

EXAMINATION OF THE URINE.

The advances made in physiologic chemistry, combined with the advances made in microscopic technique, have enhanced the ability to interpret the changes and variations which occur in the urine both with the physiologic and metabolic processes of the body, as well as in the pathologic digressions to which it is subject, to a degree little short of certainty. The uninterrupted constancy of the secretion and the ease with which it may at any time be obtained, offer us an unexcelled index of the metabolic changes going on within the economy, for it may be said that no pathologic process can seriously disturb the normal cell metabolism without producing a change of some character in the urine, which prëeminently represents the result of that process, and more quickly than any other product indicates a disturbance of normal function. Hence the application of urinalysis to the recognition of disease.

THE URINE.

Quantity. The average quantity of urine for healthy individuals is 1500 c.c., or about fifty ounces, for the twenty-four hours. In order to determine the per diem amount the patient should be provided with a clean receptacle which can be tightly covered, capable of holding the entire quantity. He must be instructed to pass the urine directly into the container and to void it before each visit to stool. A half-gallon bottle is a convenient receptacle. The quantity is diminished by perspiration and exhalations of moisture and increased activity of the bowels. Cold and dampness increase it; hence it is somewhat greater in winter. Great muscular activity, imbibition of large quantities of liquids and over-eating increase the amount; while fasting and abstemiousness diminish it.

The amount secreted is greatest after mid-day and least during sleep. The urine is increased in disease—in diabetes, chronic interstitial nephritis and amyloid degeneration of the kidney, and in those

forms of cardiac hypertrophy which are accompanied by increased intravascular pressure. Temporary abundance succeeds attacks of hysteria and other forms of convulsions.

The urine is decreased in acute Bright's disease, parenchymatous nephritis, cirrhosis of the liver, all acute fevers and inflammations and in active and passive hyperæmia of the kidney, as in early Bright's disease. The urine may be decidedly lessened or even suppressed in very acute inflammations of the kidneys, by obstruction of the ureters, by great internal injuries, and in profound shock or collapse, in the algid stage of cholera and in cases of pronounced poison of infective disease. Complete suppression can not long exist without being followed by uræmia and death.

Before deciding upon the clinical significance of increase or diminution of the volume of urine voided, careful inquiry covering the above points referable to diet and habits should be made.

Composition of the urine. The normal urinary constituents are derived from the waste products of the liquids and solids ingested and from the result of retrograde tissue metamorphosis. The urine is chiefly a solution of urea and certain organic and inorganic salts, holding in suspension epithelial cells and mucus. Their relative amounts are given in the following table taken from Parke's schedule:

Amount of urinary constituents passed in twenty-four hours.

Constituents.	*Weight 66 kilos.* Grammes.
Water	1500.00
Total solids	72.00
Urea	33.18
Uric acid	0.55
Hippuric acid	0.40
Creatinin	0.91
Pigment and other organic matters	10.00
Sulphuric acid	2.01
Phosphoric acid	3.16
Chlorine	7-8.00
Ammonia	0.77
Potassium	2.50
Sodium	11.09
Calcium	0.26
Magnesium	0.21

Each 1,000 grains of urine contain about 33 grains of solids, of which amount 25 grains are organic and 8 to 10 grains inorganic.

Changes produced by standing. On standing certain changes are brought about in the composition of the urine. The rapidity of these changes depends on the temperature (being produced much quicker at high temperatures), the accessibility of micro-organisms and the composition of the urine as voided, highly-acid urine withstanding the changes longest. When normal acid urine is allowed to stand in a tall, conical glass, there appears after some hours a floating cloud which settles near the bottom, composed of mucus in which is entangled a few bladder epithelial cells. In a cold room this occurs within two hours. It is named the nubecula. The next substance to be deposited is the reddish or yellowish alkaline urates, sodium, potassium and ammonium, and in cold urine the urates of the alkaline earths, calcium and magnesium. The latter are held in solution in warm urine. The next deposit to occur is uric acid, which crystals are dark-red or brown in color and which in part replaces the urates. At the same time or a little later form octahedral or envelope crystals of calcium oxalate. Still later normal urine deposits a sediment composed of phosphates of the alkaline earths—granular, amorphous calcium phosphate and triangular prismatic crystals of triple phosphate (ammonio-magnesium phosphate)—often interspersed with dark spiculated spherules of ammonium urate (Purdy). Upon standing for a few days all urines become turbid, owing to the swarms of bacteria which appear in the solution. Such turbidity cannot be removed by simple filtration, but may be cleared by repeated shakings with powdered charcoal and refiltering.

After a varying time, depending on the factors named, the urea is transformed into carbonate of ammonia and the normal acid reaction is changed to alkaline. The change may come about in forty-eight hours or not until after the lapse of several days. This change is spoken of as *ammoniacal decomposition* and is due to the activity of certain micro-fungi, as first pointed out by Wormley. By the same process amorphous urates are transformed into ammonium urate, uric acid is transposed into triple phosphate, and calcium phosphate is deposited.

Acid fermentation with the production of acetic and lactic acid is due to the action of contained mucus. Uric acid and the urates are

precipitated by the process and the acidity diminished. It is not common.

Color. The normal color of the urine is clear-amber or straw-color, due to the presence of urobilin or urochrom and uroxanthin. Abnormal coloring is due to blood, bile, melanin, hæmoglobin, vegetable colorings and those derived from medicinal sources. Imbibing large quantities of water renders it paler, and abstinence and strong diet by concentration increase the color. As a rule both the depth of color and the acidity increase with the specific gravity and the increase of the solid constituents, and vice versa.

In disease the urine is deficient in coloring matter when abundant, as in diabetes, hysteria, interstitial nephritis and amyloid kidney. In the first it may have a pale-green cast when viewed with transmitted light. Fevers and diseases which concentrate the secretion increase both relatively and absolutely the amount of coloring substances. The presence of blood and hæmoglobin products produces a red or brown color. Bile pigment gives the urine a dirty-greenish tint. Diabetic urine may also show a faint-greenish tinge. Carbolic acid and creosote color the fluid dark-brown or black, santonin imparts a yellow color.

Odor. The odor of fresh urine varies with the degree of concentration; sweetish, aromatic, characteristic, it has given rise to the adjective "urinous." The odor is due to acid derivatives of the phenyl group. Stale urine is ammoniacal. Putrid urine is due to the decomposition of mucus and other organic substances. Foods and drugs influence the odor. Asparagus, garlic, cabbage, impart a well-recognized odor, and the essential and aromatic oils and turpentine give character to the odor of the urine. Cystitis gives rise to ammoniacal urine (Nothnagel says acid). Pyuria has the odor of pus or of decomposition. The odor of acetone is often noticeable in the urine of eclampsia and of diabetes.

Reaction. The reaction of fresh normal urine is acid, due to acid sodium phosphate. The acidity increases for several hours after being voided, due to the so-called acid fermentation. During this period a whitish deposit, sometimes pink or red, of acid urate of sodium or uric acid is deposited, which disappears on warming. The acidity is least between meals; early morning urine may be alkaline. Alkalinity due to ammonia (inflammatory alkalinity) causes red litmus to turn blue when moistened therewith, but to resume its original color when

PLATE XXIV.

VOGEL'S SCALE OF URINE TINTS.

dry. The urine is rendered alkaline by the administration of alkalies, as soda or potash, the alkaline carbonates or the salts of the vegetable acids, hence a vegetable diet reduces acidity. Acidity is increased by administration of acids, by a meat-diet and by concentration.

Specific Gravity. The normal specific gravity of urine varies from 1018 to 1022, the average may be placed at 1020, but varies with food and drink. Meat-diet, active exercise, copious sweating raise it above this figure. Fluids imbibed lower it. Those forms of Bright's

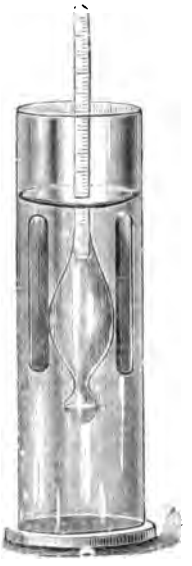

Fig. 71.—Squibb's Urinometer.

disease with increased urine are accompanied by diminished specific gravity, while the acute variety and diseases which diminish or concentrate the secretion raise the figures. When in disease the volume of the urine remains unchanged and the specific gravity falls, it means faulty elimination and its import is unfavorable. The presence of sugar raises the specific gravity, and urine of a specific gravity of 1028 or over should be examined therefor. Squibb's urinometer is recommended for office use. Corrections for temperature in taking the specific gravity are sometimes necessary in very hot or cold weather. The rule is to add one degree of specific gravity for every 10° F. of the urine above the degree at which your urinometer is standardized

(Squibb's at 77° F.) and to subtract one degree for each 10° F. below standard.

In using the instrument the bulb of the urinometer should be first completely immersed and care should be taken that it does not rest in contact with the side of the container.

Transparency. Fresh healthy urine is clear. If urine is turbid when passed it is pathologic. Mucus renders it cloudy. This cloudiness is unaffected by heat, mineral acids or alkalies, but is increased by the addition of acetic acid. Precipitates of urates, carbonates and phosphates render it cloudy. Urates redissolve on the application of heat, phosphates disappear on the addition of mineral acids. In fevers, the quantity of urine is occasionally so small that the urates separate out before the urine leaves the bladder. This is not infrequently the case in pneumonias and in capillary bronchitis of children, owing to deficient oxydation. Increase of turbidity on the application of heat is due to organic substances, albumin, pus or blood, or to earthy phosphates. Increased turbidity on the addition of mineral acids is due to organic elements.

The amount of solids in the urine is obtained approximately by multiplying the last two figures of the specific gravity by 2.33. The product equals the number of grams per 1,000 c. c. of urine. This number is known as Häser's coëfficient. By multiplying by 0.233 the percentage of total solids is at once obtained. For children the coëfficient is too high, 1.80 should be used.

The average excretion of solids in health for adults is sixty grams per diem, but decreases about 10 per cent. for each decade after forty. Decided decrease points to renal insufficiency, and in nephritis portends uræmia. In the course of any acute disease, the elimination of solids should vary with the temperature, since high temperature means active tissue metamorphosis, hence in these conditions decrease in solids means faulty elimination.

Of the urinary solids, urea constitutes the greatest proportion, being normally from twenty to forty grams in the 1500 c. c. per diem urine, with an average of thirty-three grams. Urea represents the ultimate oxydation of nitrogen in the organism.

Acute sthenic conditions, mental and muscular activity and nitrogenous foods increase the output of urea. Chronic diseases, asthenic diseases, Bright's disease, and diseases of the liver retard the elimination of urea.

Detection of Urea. Place a few drops of the urine on a glass slide and add half as many drops of nitric acid. On warming, rhombic and hexagonal crystals of nitrate of urea are formed and recognized under the microscope. Fluids which do not give this reaction are not urine,—a noteworthy point.

Quantitative determination of urea requires practice. The ureometer of Doremus is sufficiently accurate. The reaction is due to the fact that when a solution of urea is brought into contact with a solution of sodium hypobromite, the urea undergoes decomposition

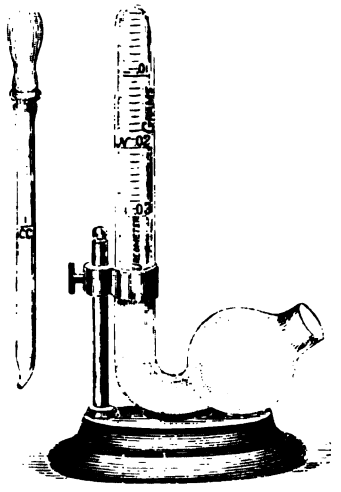

Fig. 72—Doremus' Ureometer and Pipette.

with the freeing of all its nitrogen. Thus: $CON_2H_4+3NaBrO=CO_2+3NaBr+2H_2O+N_2$.

The long arm of the tube is filled to the bend with the sodium hypobromite solution by inverting, and the instrument righted. Then, with a graduated pipette one c. c. of urine is introduced and slowly discharged beneath the surface of the solution. The nitrogen set free rises and the displaced mixture flows into the bulb. After ten minutes the reading is taken. The scale reads in milligrams, which indicates the amount per c. c. of urine. Normal urine gives .02 gm. per c. c. which is two per cent. Another form of scale gives the percentage or grains per fluid ounce.

The hypobromite solution must be freshly prepared as follows: Dissolve 100 grams of sodium hydroxid in 250 c. c. water. For use,

take 10 c. c. of this solution and add 1 c. c. of bromine, mix and dilute with 10 c. c. water. This amount is sufficient for one test.

Owing to the dangers, the instability and the disagreeable properties of bromine, I prefer the method of Bartley. A straight tube, closed at one end, graduated so as to give the number of grains per fluid ounce, a pipette holding one c. c. and the ordinary urine pipette are required. The tube is filled to the fifth division with a twenty per cent. solution of KBr. Chlorinated soda solution, NaClO (Labarraque's), to the fifteenth or twentieth division is added. A small

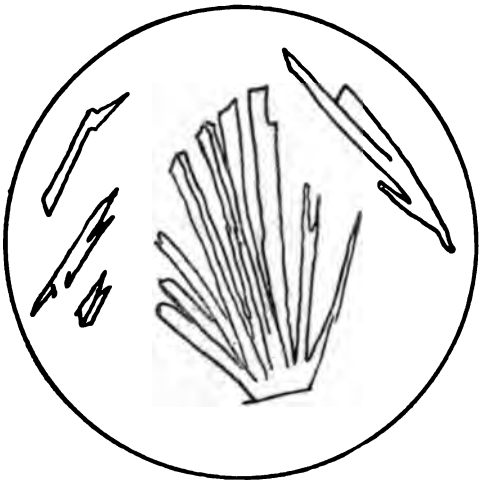

Fig. 73—Urea from aqueous solution.

quantity of pure water is now floated upon the top of these reagents by inclining the tube and adding the water from the pipette. One c c. of urine is now carefully floated upon the water by the same method. Tightly closing the open end with the thumb the tube is inverted and shaken. After effervescence ceases the surface reading of the fluid is noted. Still closed, the tube is plunged into a pail of water and the thumb removed. The imprisoned liquid falls and the tube is immersed until the outer and inner liquids are at the same level, and the reading again noted. The difference in the two readings is the number of grains of urea in one fluid ounce of urine, which, multiplied by the number of ounces passed in twenty-four hours, gives the number of grains per day. A quantity of less than 350 grains per day should be regarded as pathologic.

Uric acid is readily recognized by the microscope. Its various crystals are shown in the figure. If urine be kept until ammoniacal decomposition occurs, uric acid crystals deposit. In general, conditions which increase urea increase uric acid.

Dyspnœa and impeded respiration increase it. Bright's disease and gout diminish its excretion. The normal amount is 0.6 to 0.8 gms. per day. It is determined by filtering a known quantity of urine and adding 5 c. c. of HCl to each 100 c. c. After twenty-four hours collect the precipitate on a previously-weighed filter paper, wash, dry, weigh and deduct the weight of paper. The result is the weight of uric acid in the amount of urine employed.

Ethereal sulphates in excess in the urine indicate putrefactive changes of grave order, within or outside the digestive tract, and the increase is proportional to the severity of the process.

Phenol potassium sulphate and indican are representatives of this group. Both indoxyl-potassium sulphate and indolyl-sulphuric acid are known as indican, and respond to the same tests. Excess of this substance indicates putrefaction of albumin with absorption of indol. It especially points to obstruction of the small intestine. In purulent pleurisy and peritonitis it occurs in abundance. It is increased in wasting diseases and starvation, hence is found in phthisis and cancer, likewise in dysentery, typhoid fever and acute brain diseases. In health the urine contains a mere trace of the ethereal sulphates.

Detection. Mix equal quantities of urine and strong HCl in a test tube, add drop by drop a freshly made saturated solution of chlorinated lime (bleaching powder) or chlorine water, until the solution fails to darken on further addition. Shake the blue solution with chloroform which takes up the indican and indicates by the depth of its color, the amount present.

Inorganic Constituents. The chlorides are the principal inorganic constituent of the urine and the quantity excreted averages 10 to 15 grams daily, thus ranking next to urea. The excretion of the chlorides is lessened in all acute febrile diseases, in dropsies and serous exudations. In grave cases of pneumonia they may disappear from the urine. In rheumatism their sudden disappearance should arouse the suspicion of endocarditis or pericarditis. Their reappearance or increase is favorable from a prognostic point in all these conditions.

The chlorides are precipitated by solutions of nitrate of silver.

In most cases an approximate estimate of their quantity is all that is necessary to the clinician. I have used the following:

Add to a volume of urine a few drops of nitric acid, then a solution of $AgNO_3$ (1 to 20). The chloride of silver falls as a white, curdy precipitate which should occupy one-fourth of the volume of the urine taken. If the precipitate, after standing, occupies much more or less than this volume the chlorides are increased or diminished. A counter-test with normal urine always should be made for comparison. The silver nitrate solution should be added, drop by drop, until its further addition fails to produce a precipitate.

Ultzmann's method: A standard solution of nitrate of silver, one drachm to the ounce, is prepared. To half-a-glass of urine add a few drops of nitric acid, then one or two drops of the silver solution. "If a white, flaky precipitate occurs, quickly sinking to the bottom without diffusing, the chlorides are undiminished. If simple cloudiness, without curdy flakes, appears and readily diffuses, the chlorides are diminished to 0.1 per cent. (normal, 0.5 to 1.0 per cent.). Should no precipitate occur the chlorides are absent."

Phosphates. The amount of phosphoric acid in the day's urine varies between 2.5 and 3.5 grams, excreted as alkaline and earthy phosphates.

The earthy phosphates are insoluble in alkaline urine and on heating the precipitate is increased and may be mistaken for albumin. They disappear on the addition of acid, while albumin remains. Triple phosphates are not present in normal urine when voided, but form on standing. In cystitic urine they are found. The acidity of the urine depends upon the presence of acid sodium phosphate. Acute fevers, gout, most kidney diseases and pregnancy diminish the phosphates. Wastings, especially osteo-malacia and rickets increase their excretion.

The earthy phosphates are precipitated by rendering the urine alkaline and heating.

The alkaline phosphates are precipitated by magnesium mixture, which is composed of $MgSO_4$ and NH_4Cl, each one part; aq. ammonia, one part; water, eight parts.

The sulphates in the urine increase or diminish in general with the urea.

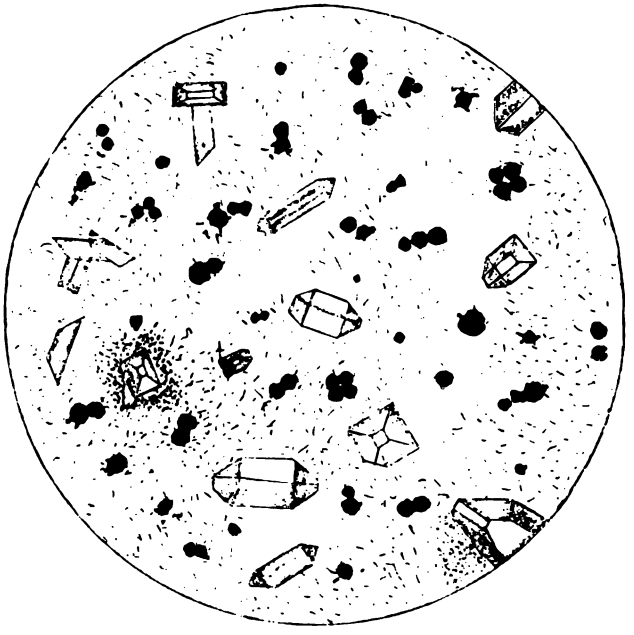

PLATE XXV.

Triple phosphate; ammonium urate; bacteria.
Alkaline fermentation.

ABNORMAL CONSTITUENTS OF THE URINE.

Albumin. Of all the abnormal constituents of the urine, from a clinical standpoint, the greatest interest centers upon albumin and its significance. Briefly summarized, its presence means alteration in the structure of the kidney, alteration in the composition of the blood or of blood pressure.

1. *Albuminuria without coarse renal lesions* is a matter of dispute. After cold-bathing, muscular exercise, dyspepsia, hysteria and violent emotions, it is found in the urine occasionally, and the changes which permit the transudate are certainly not permanent. Cyclic or adolescent albuminuria is interesting. Boys are oftenest the subjects and recovery follows after a varying period.

After sixty, it is not uncommon to encounter small quantities of albumin in the urine, associated with mucin, and the tendency increases with each decade thereafter.

2. *Albuminuria of pyrexia.* Albumin may be present in fevers of almost any degree, the lesions which cause it generally passing away with the disappearance of the primary disease. It is seen in tonsillitis, typhoid fever, pneumonia, malaria, the entire group of specific infectious diseases which used to be called zymotic, cholera, yellow fever and other infections.

3. *Albuminuria due to blood changes.* All profound anæmias, leukæmia, purpura, syphilis, scurvy, rickets, poisoning by metallic substances as arsenic, lead, mercury or phosphorus, are followed by albuminuria which in some cases is transient, in others leads to permanent lesions. The administration of anæsthetics is followed in rare cases by albumin. Certain states and conditions also manifest transient albuminuria, as the puerperal state, exophthalmic goitre, tetanic seizures, after apoplexy and epileptic fits, purpura and hospital gangrene.

4. *Albuminuria with kidney lesions.* Here the transudate occurs in states of active congestion, as in inflammations of the kidneys; in passive congestion due to obstruction or pressure, or such as occurs in organic heart and lung diseases. Organic lesions of the kidneys, constituting the entire group of Bright's diseases, the degenerations and suppurative processes of the organs and growths within their structure, constitute the chief causes of albuminuria.

5. *Pus-producing inflammations* of any part of the urinary tract are accompanied by albumin in the urine.

6. *Albuminuria accompanying impediments to the circulation,* in which the kidneys are not primarily involved, as tumors, abdominal growths, cirrhosis and other changes in the liver.

The amount of albumin cannot be taken as indicative of the gravity of the lesion, since in some serious alterations, such as interstitial nephritis, the amount is small and intermittent: nor can albumin without other evidences of renal alteration be accepted as indicative of organic disease, but in general, its increase or decrease in established recognized conditions, indicates the gravity of the causative disease.

Tests for Albumin. Typical albuminous urine is of low specific gravity, pale greenish-yellow in color, and forms a permanent froth when shaken. Soon after being passed a sediment is deposited.

The potassium ferro-cyanide test is one of the easiest and most reliable tests at command. I have found it most satisfactory. It may be applied as follows: A quantity of urine is placed in a test tube, half as much of a solution of potassium ferro-cyanide of a strength of 1 to 10 is added and the tube shaken. On the addition of a few drops of acetic acid, albumin, if present in any form, is precipitated in a flocculent cloud. The test gives no reaction with mucin if the acetic acid be added *last*, and no reaction with phosphates or urates, hence any precipitate which occurs is albumin, albumose or nucleo-albumin.

Another method of applying the test is to add acetic acid first and filter out the precipitated mucin, then float the clear acidified urine upon the top of the ferro-cyanide solution. A white zone forming at the point of contact indicates albumin.

Heller's test. This test is probably more universally used than any other test and is reliable to those familiar with its variations. A test tube is filled to the depth of one inch with pure nitric acid. A long pipette is filled with urine, which is floated upon the surface of the acid by considerably inclining the tube and slowly discharging the urine against its side. The presence of albumin of any kind is indicated by a white zone at *the point of contact*. The width varies with the amount of albumin present.

The amorphous urates when precipitated by this method appear as a brown cloud or zone in the urine, above, and not *at* the point of contact, and can hardly be mistaken for albumin. Their density diminishes upwards. Mucin may show as a cloud towards the top of

the tube. The only probable error is in patients who have been taking balsams, in which case the precipitate resembles albumin, but is cleared by addition of a small quantity of alcohol. While the precipitate generally appears immediately, yet when very scanty may require a few minutes to form, hence the tube should be re-examined at the end of half an hour.

Boiling the urine in a tube coagulates albumin, and also precipitates the earth phosphates. The latter redissolve on the addition of nitric acid, which should be added drop-by-drop as the urine is reboiled. Albumin in small quantity is apt to escape detection by this test and excess of phosphates may deceive the observer. The test is reliable for albumin in considerable quantity, but is inferior to either of the other tests given and less easy of manipulation.

Mucin. Mucin is the secretion of the mucous cells. It is a gluco-proteid, i. e. a compound of a proteid and a carbo-hydrate and is closely related to the albumins, hence constitutes a fruitful source of error in searching for small quantities of albumin, especially if the amount of mucin is increased. It is present in all urine and in inflammatory conditions of the urinary tract is increased. In catarrhal inflammations of the pelvis, of the kidney or the bladder the amount may be enormous. Mucin is increased in febrile processes and is sometimes the forerunner of albumin in these cases.

It is not coagulable by heat and is soluble in strong mineral acids and in alkalies, but is precipitated by acetic acid, alum and very dilute mineral acids, which latter property gives rise to the propensity to mistake it for albumin. Mucin is insoluble in alcohol, ether, or chloroform. Owing to its solutions dissolving oxide of copper, the presence of mucin in abundance hinders the test for sugar.

Detection: Dilute the urine to double its volume and add excess of acetic acid. The mucin is precipitated.

Pus. Pus renders the urine turbid to the naked eye. It quickly deposits as a whitish or greenish sediment. The supernatant liquid contains albumin and globulin. Heat does not dissipate the sediment, hence distinguishes it from urates. Acids do not dissolve it as they do the earthy phosphates. The addition of strong alkaline solutions dissolves it with the production of viscid or ropy masses (Donné's test). The addition of hydrogen dioxide causes rapid effervescence,—a valuable test for pus in any fluid.

The microscope shows characteristic cells.

CARBOHYDRATES.

While glucose, levulose, inosite and lactose may occur in the urine, the most important clinically is glucose. Whether or not glucose is a constituent of normal urine is still disputed. Very delicate tests frequently demonstrate it in urine otherwise normal. When glucose appears in the urine in appreciable amount it is known as *glycosuria*.

Glycosuria may appear as a temporary condition in various diseases of the brain and cord, lungs, liver, heart, during pregnancy and

Fig. 74—Lipogenic Glycosuria. Urine also contains albumin.

in certain infectious diseases, as diphtheria, influenza, rheumatism, typhoid fever, syphilis, scarlet fever, cholera and cerebro-spinal meningitis.

It is often encountered in obese persons, particularly Hebrews, and is known as lipogenic glycosuria. Certain drugs and toxic materials produce a substance which gives to the urine a similar reaction, as morphia, chloral, hydrocyanic acid and amyl nitrite. Both temporary and permanent glycosuria have followed the inhalation of nitrous oxide. The internal use of turpentine, of salicylic acid, mercury, alcohol and a few other substances is sometimes followed by the reaction. Phloridzin, and some of the other substances mentioned, produce glycosuria by directly attacking the renal epithelium and destroying its power to keep back glucose. The ingestion of a larger

quantity of carbo-hydrates and peptones than the liver can take care of, causes a temporary glycosuria.

Persistent glycosuria is known as *diabetes*. It is not a disease but a symptom and the causative lesion must be looked for in the liver; the pancreas—which shows lesions in fifty per cent; the nervous system—where several lesions have been found in connection with cases, but none typical; or in the kidneys. The heart, the lungs and other organs show frequent lesions which are all probably secondary.

Diabetic urine is generally pale straw-color, often with a greenish tint, has a characteristic, sweetish odor and taste, and a specific gravity varying between 1030 and 1050. The amount of sugar present varies from two to twelve per cent. The quantity of urine is much increased, being seldom less than 1600 c. c. and may reach 8000 c. c. per diem. The gravity of the disease increases with the polyuria. Glycosuria without polyuria is seldom fatal, especially in advanced life. The severity of the disease in all cases bears a relationship to age. In the young it is progressive and almost uniformly fatal, running its course in a few months to a few years. After thirty-five the disease progresses slowly and the prognosis is better. After fifty, it is often amenable to treatment.

Tests for glucose. The methods of detection most often used are by means of the copper tests, which depend upon the fact that solutions of grape sugar in the presence of an alkali reduce cupric oxide to lower oxides. The presence of albumin interferes more or less with the tests and, as it is easily removed by boiling and filtering, it is wise to do so.

Trommer's test. Add to a quantity of urine in a test tube about half its volume of sodium or potassium hydroxide. Then add, drop by drop, a solution of copper sulphate (1 to 8) until a slight bluish-white precipitate is formed. The precipitate is cupric hydroxide. In the presence of glucose it is dissolved on shaking and a dark-blue color results. On heating, if glucose be present, red cuprous oxide and yellow cuprous hydroxide fall, just as the boiling point is reached. If, instead of boiling, the tube is allowed to stand for half an hour the same reduction occurs. With heat the test is extremely delicate. If no precipitate occurs, sugar is absent.

Precautions. If sugar is present in considerable quantity, the precipitate forms without boiling. The urine should not be kept boiling, *but merely brought to the boiling point,* since continued boiling causes

other substances to reduce the copper, as uric acid, creatin, mucus, albumin, peptones, hypoxanthin, creatinin, glycuronic acid, carbolic acid, certain alkaloids and excess of coloring matter. As a rule, these give a greenish or greenish-yellow, not a red, deposit.

Precipitate of phosphates may occur on the addition of the hydroxide, but is white and flocculent and bears no resemblance to the red, granular cuprous oxide. A yellow precipitate, which falls on cooling, is cuprous oxide and is not due to glucose. A red granular precipitate is very probably sugar.

Haine's test. This is a modification of Fehling's test. Formula: Pure copper sulphate, 30 grains; distilled water, ½ ounce, dissolve; add pure glycerine, ½ ounce; mix and add 5 ounces liquor potassæ. The solution is somewhat more stable than Fehling's solution.

Method: Boil one drachm of the test fluid in a tube and note whether it remains clear. Add six to eight drops of urine and reboil. A copious red or yellow precipitate indicates sugar. If no precipitate occurs, sugar is absent. Here again the alkali may precipitate the earthy phosphates, but they should not be mistaken for sugar. The precautions given under Trommer's test should be observed.

Fehling's test is applied in the same manner as Haine's. The quantity of urine added should never exceed the volume of the test solution used. Fehling's solution is best prepared in two parts.

I.—Dissolve 34.64 grams of crystallized $CuSO_4$ in water and dilute to 500 c. c.

II.—173 grams Rochelle salt and 60 grams NaOH are dissolved in water and diluted to 500 c. c.

For use, mix equal volumes. This solution is also used for quantitative examination, 10 c. c. of the mixed solution equals 0.05 grams glucose.

Phenylhydrazin test. When this substance is boiled with grape sugar a characteristic crystalline compound results.

Test: To 50 c. c. of suspected urine add 2 gms. phenylhydrazin hydrochloride, and 2 gms. sodium acetate, 10 c. c. of water may be added to promote solution. Dissolve, and heat on water-bath for one hour. On cooling, a yellow deposit forms, a few drops of which is placed on a glass slide. Bright, needle-like crystals of phenylglucosazone are formed, rarely invisible to the naked eye. Scales and brown granules are not indications of sugar. The microscope shows radiating acicular structure. The crystals indicate that the urine contained

a carbo-hydrate and the test is therefore reliable. None of the substances mentioned under Trommer's test is capable of producing the reaction.

Quantitative Determination of Sugar. The amount of sugar in the urine indicates the severity of the disease and is the basis of prognosis. Its increase or diminution is the best evidence of the result of treatment.

Roberts' differential test is accurate and simple, but requires twenty-four hours for its determination.

Method: Place 4 ounces urine in a large bottle, add ¼ cake of compressed yeast and cork with a perforated cork. The same quantity of urine is placed in another bottle and corked. Both are allowed to stand twenty-four hours. The alcohol and carbonic anhydride formed by the fermentation reduce the specific gravity one degree for each grain of sugar per fl. oz. of urine. Thus, if the sp. gr. is reduced from 1040 to 1030, the urine contains ten grains of sugar per ounce.

When fermentation is completed the urines are decanted and the sp. gr. of each separately obtained. The number of degrees of density lost in the fermented urine indicates the amount of sugar per ounce, or, the number of degrees lost multiplied by 0.23 gives the percentage of glucose. The results are approximately accurate.

Fehling's test is the most satisfactory yet devised. The only objection that can be urged is that practice is necessary to determine the precise point of reduction.

Method: 10 c. c. of Fehling's solution and 30 c. c. water are measured into a porcelain dish and kept at the boiling point over a Bunsen burner or alcohol lamp. The urine is added, drop by drop, from a burette until the blue color of the Fehling's solution has entirely disappeared. The number of c. c. of urine required to produce this result is noted and the sugar calculated thus: It requires 0.05 gm. of sugar to remove the coloration of the ten c. c. of solution used, hence the number of c. c. of urine dropped contained 0.05 gm. of sugar. Say 2 c. c. of urine were used, then 100 c. c. which is 50×2 contained 50×0.05 or 2.5 grams per 100 c. c. By dividing 5 by the number of c. c. of urine used the result is the same, and the per cent. thus easily obtained.

When considerable sugar is present the urine should be diluted with a known volume of water, as one to five, and the result corrected in accordance with the dilution.

Acetonuria. Whether acetone occurs in healthy urine or not is disputed. It occurs after alcoholic indulgence, over-eating of proteids and in the urine of children. It occurs in fevers and rises and falls with the temperature curve. It is often associated with carcinoma and should be sought for in suspected cases. In diabetes its appearance often precedes diaceturia. It is capable of producing a species of auto-intoxication, and often occurs in the insanities.

Chautard's test is the simplest. An aqueous solution of magenta is decolorized with sulphurous acid. A drop of this solution added to fluids containing acetone gives a violet color. When the amount of acetone is very minute, the color develops after five minutes.

Diaceturia. Ethyl-diacetic acid in the urine is of serious import. It occurs under the same febrile conditions as acetone. In diabetes it is the premonitor of coma and signifies death. It occurs oftenest in young subjects. According to von Jaksch, the cause of coma in diabetes is diacetic acid.

Detection: Boil a sample of the urine and add a few drops of ferric chloride. A deep-red color is produced. If the phosphates are precipitated, add the chloride until precipitation ceases, filter and repeat the test. No other substance gives rise to the red color in the boiled urine.

BILE.

Bile acids and bile salts appear in the urine during attacks of jaundice. Increase of bile salts occurs with the termination of "bilious attacks." They are increased in many forms of organic disease of the liver, in splenic leucocythæmia and anæmia. Bile pigments are found in the urine in phosphorous poisoning, in grave disorders of the liver, in obstructive jaundice, where the coloring matter reaches the urine through the circulation, and precedes its appearance on the skin. Both may appear during excessive summer heat and after the use of alcohol. The color of the urine is yellow, green, brown or black. On shaking it yields a yellow froth which is characteristic and in the absence of ingested drugs such as rhubarb, chrysorobin and the like, is distinctive.

Gmelin's test for Bile Pigment. A small quantity of commercial nitric acid (yellow nitrous acid) is placed in a test tube and the urine is floated upon it. A rainbow coloration appears at the point of contact. Or the urine may be passed through filter paper, and a drop of

the acid placed in the center of the wet paper, when the colored rings appear.

THE DIAZO-REACTION OF EHRLICH.

This test was proposed by Ehrlich in 1882 as a valuable diagnostic sign of typhoid fever. Although the reaction occurs in a few other diseases, as pulmonary tuberculosis, yet in the cases most often mistaken for typhoid fever the reaction does not occur, viz., malaria. I studied the urine in 36 cases of typhoid fever in soldiers returned from Tampa. The reaction was pronounced in 35. It was absent in all cases of malaria when typhoid was absent.

It occurs from the fourth to the seventh day and thereafter. The intensity of the reaction varies with the gravity of the case. When absent, the diagnosis is doubtful. It occurs in rapidly fatal cases of phthisis, sometimes in measles, miliary tuberculosis, pyæmia, scarlet fever and erysipelas. It is absent in apyrexial disease (Ehrlich). Two solutions are prepared:

I.—2 grams of sulphanilic acid; 50 c. c. HCl; 1,000 c. c. water. Mix.

II.—A 0.5 per cent. solution of sodium nitrite.

Method: 50 parts of No. I and one part of No. II are placed in a test tube, an equal quantity of urine added, and the solutions well mixed. About $\frac{1}{8}$ of the volume of ammonia is then added. If the reaction is positive a carmine or cherry-red color develops, and on shaking the color extends to the foam. Von Jaksch thinks the reaction due to acetone and not especially significant of typhoid.

URINARY SEDIMENT.

When urine is allowed to stand for some hours gravity causes it to separate into two layers, the solid portions or sediment and the supernatant liquid. The solids consist of insoluble materials contained in the fluid and soluble substances which by chemical activity or crystallization have been separated from the solution. Much time is saved and many difficulties are overcome by separating the urinary sediment immediately by means of the centrifuge, instead of waiting for gravity to accomplish the same end.

We have already noted some of the changes which take place in normal urine on standing. Changes more rapid and more radical occur in pathologic urine. Much more information is therefore

gained by an immediate examination of such urine, before casts have macerated and soluble substances which may have been passed as solids have had time to dissolve, and before pernicious bacterial activity has had time to destroy contained elements or to form new ones. In the same way crystalline substances which are passed in a state of crystallization may be distinguished from those which form upon standing. By the centrifuge the elements are more completely separated and concentrated, hence such as occur in only minute quantity are less likely to escape detection than when collected by gravity.

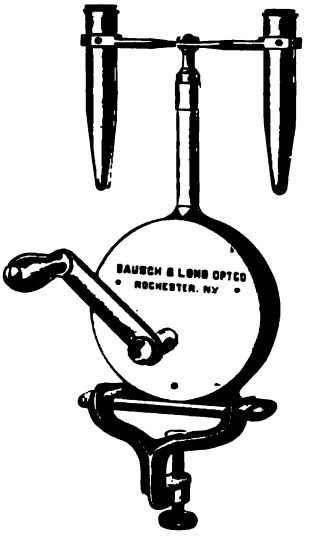

Fig. 75—Hand Centrifuge.

It is difficult to satisfactorily classify the two groups of substances which make up the urinary sediment. Organized and unorganized are terms frequently used, but unorganized does not in this case mean non-organic and is misleading. Perhaps the division into *chemic* and *histologic* sediments is as useful as any.

The *chemic substances* are uric acid, the urates, phosphates, calcium oxalate, cystin, leucin, tyrosin, melanin.

The sediments of acid and alkaline urine differ markedly. Acid urine may deposit: (a) uric acid; (b) acid urates of sodium, potassium, ammonium and calcium; (c) calcium oxalate; and occasionally, (d) hippuric acid; (e) calcium sulphate, cystine, leucin, tyrosin.

PLATE XXVI.

URIC-ACID CRYSTALS. (Natural color.)

In such urine the uric acid and the calcium oxalate are crystalline, the urates amorphous or granular, except sodium urate which is sometimes crystalline.

Alkaline urine may deposit: (a) amorphous phosphate and carbonate of calcium; (b) crystalline urate of ammonium; (c) phosphate of calcium and magnesium; (d) triple phosphates.

The most important constituent of the alkaline sediment is the *amorphous earthy phosphates* which are regularly precipitated in urine that is alkaline when voided, as well as in urine that has undergone fermentation. The crystalline forms are the triple phosphates, ammonium urate, calcium phosphate and carbonate, and, under pathologic conditions, leucin, tyrosin and cystin.

Uric acid crystals occur as a deposit only in acid urine. They are deep-red or yellow in color, prone to cling to the sides of the vessel and can be recognized by the trained eye. Cubes, plates and rhombic crystals, alone or coalesced into beautiful stellates and ribbon bow-knots, are found and are easily recognized under the lens by color and form. Threads suspended in urine attract the crystals and their tendency to aggregate around any suitable nucleus explains the formation of uric acid calculi, the most frequent of all forms of gravel. Uric acid may be said to be pathologic when it is deposited shortly after the urine is passed. Occurring in urine which has stood ten hours or more, it has little clinical significance. Excess of uric acid, high acidity, diminished mineral salts and pigments, favor its deposit. Over-indulgence in animal food produces the first two of these factors. Fever favors its production by diminution of the liquid elements and raising the acidity of the urine.

Acid urates of sodium, potassium and ammonium, sometimes of calcium, occur as amorphous or crystalline deposits. The sodium salt occurs in the brick-dust sediment so often seen in winter urine. It occurs as amorphous granules or stellate or fan-like crystalline clusters. Its color is from pink to brown, according to the amount of urinary pigment present. The calcium and potassium forms are amorphous.

Ammonium urate occurs as coarse, spherical masses studded with spicules. The colors are yellow to deep-brown and the crystals are known as "hedge-hog" or "thorn apple" crystals, easily recognized. They are more apt to be found in alkaline urine.

The above form the mixed urate sediment so often seen on

the sides and bottom of the vessel. The deposit is increased by fever, wasting disease, organic disease of the liver, dyspepsia and gout. It is recognized by its disappearance on the application of heat.

Oxalate of lime occurs in either acid or alkaline urine. It is oftenest seen associated with triple phosphates. The crystals are small, highly refracting octahedra or envelope crystals or "dumb-bell" crystals and are unmistakable under the microscope. They disappear on the addition of HCl. Calcium oxalate is a constituent of certain

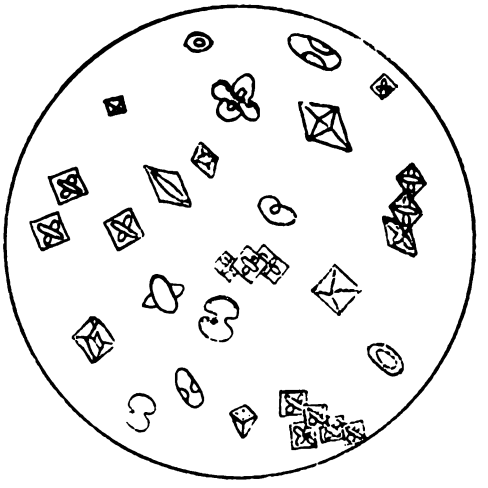

Fig. 76.—Oxalate of Calcium.

vegetables and their use increases greatly the amount in normal urine. Tomatoes, rhubarb, asparagus and grapes contain excess of the salt; "greens," cabbage, turnips and apples contain smaller amounts. Excessive meat ingestion causes excretion of large amounts of the salt.

Hippuric acid excreted in large amounts by the herbivora, is found only as a trace in normal urine. After taking benzoic acid or eating certain fruits, the crystals are seen, but have no clinical significance. They occur as colorless prisms with sharply-defined ends and as needles. They do not respond to the murexide test as does uric acid.

Calcium sulphate crystals occur as radiating needles and are of little importance.

Phosphates. The alkaline phosphates are not found in urinary

sediment. The earthy phosphates are the ammonio-magnesium phosphate, known as triple phosphate, and calcium phosphate.

The first occurs in various forms, the beveled triangular prism, or "coffin lids" being the most characteristic. These may be square or oblong, are glassy or light-green in color and easily recognized. *They disappear on the addition of acetic acid.* Stellate feathery forms are less frequent but easily distinguished.

Calcium phosphate is usually an amorphous, granular, white deposit which, as said, is precipitated by heat and may be mistaken for

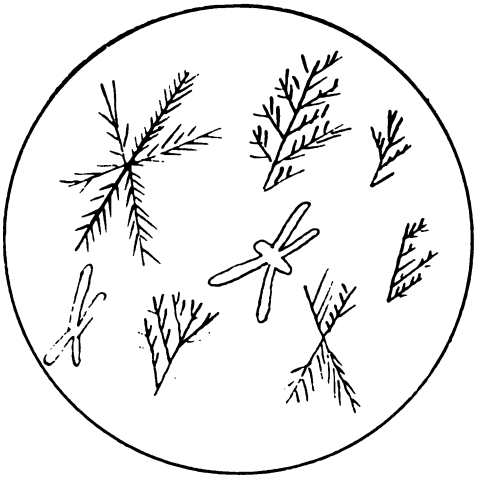

Fig. 77—Triple Phosphates, Pine Branch Crystals, Rapid precipitation.

albumin. The crystalline form, rarely seen, consists of rods, separate or grouped into wedges, or of stellate, colorless rosettes.

The deposit of triple phosphate in freshly voided urine means ammoniacal decomposition in the urinary passages, as pyelitis and cystitis. The deposit of phosphates in other urine occurs in convalescence, in dyspepsia, particularly nervous dyspepsia, phthisis and as pointed out by Sir William Roberts, in cancer.

Cystine is rarely seen. It may occur in diminished bile secretion. It occurs as opalescent hexagonal tablets which sometimes overlap each other. It aids in forming calculi. A drop of HCl or of ammonia on the slide causes its disappearance. With the latter it reappears on evaporating the reagent.

Leucin is rare. It appears as white lamellæ or as yellow spherules

like drops of oil. It resembles sodium urate, but unlike it is not dissolved by heat. From oil drops they are distinguished by not being dissolved by ether.

Tyrosin occurs with leucin in small-pox, typhus, typhoid, in acute atrophy of the liver, leucocythæmia and in phosphorus-poisoning. It occurs in yellow-green globules or as fine needle-like radiating crystals.

HISTOLOGIC SEDIMENT.

The histologic sediment found in the urine includes pus, blood, epithelium, casts, spermatozoa and bacteria. More rarely are found fragments of tissue, of new-growths, parasites or their eggs.

Fig. 78.—Triple Phosphates—Slow Precipitate, Amorphous Urates. A Stellate Forms. B Coffin lids.

Pus is the most frequent histologic constituent of the urine and may come from any part of the urinary tract. Pus-containing urine is turbid when voided and responds to the tests for albumin. Pus corpuscles are easily recognized by the microscope as pale, circular cells containing finely granular protoplasm and one to three distinct nuclei. Treatment with acetic acid causes them to lose their granular appearance and the nuclei to become distinctly visible. Their size is nearly double that of the red blood corpuscle. When a quantity of pus is treated with potassic hydrate it dissolves into a homogeneous,

sticky mass which will not flow (Donné's test). In testing allow the pus to settle, then pour off the supernatant liquid before adding the hydrate.

Pus is frequently mixed with micro-organisms and with epithelium or other tissue elements derived from the locality of its origin.

Pus derived from the kidney is intimately mixed with the urine when passed, the reaction is acid and the kidney epithelium is found, and bladder symptoms are absent. Renal epithelial cells are small, round or polygonal with a single large nucleus. Their size distinguishes them from pus cells.

Pus-urine from the bladder is alkaline when voided or quickly

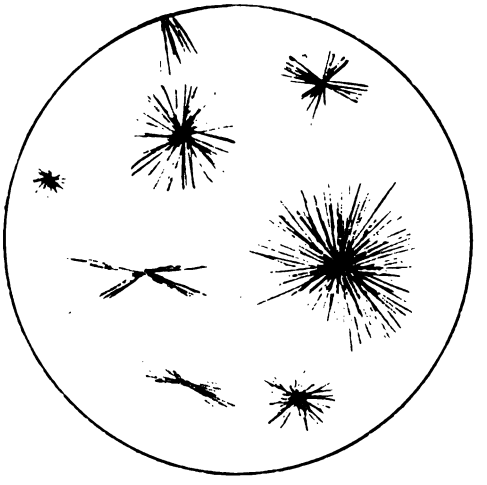

Fig. 79.—Tyrosin Crystals.

becomes so, contains liberal quantities of mucus, triple phosphates and flat, large epithelial cells from the bladder.

Pus from the urethra may be squeezed out, or flushed out with the first drops of urine. In cases of doubt, washing the urethra before micturition frees the urine from pus. In prostatic disease the pus often appears as long threads bound together with mucus.

Blood. Blood corpuscles appear in the urine under a number of pathologic conditions. Their bi-concave character and the alternate change from dark to light of rim and center, which takes place on focusing, owing to this formation, aid in their recognition. After having been soaked for some time in the urine they lose their sharp

outline, swell, pucker and resemble the pus corpuscles, but can be distinguished by the absence of nuclei. If in doubt, a small quantity of the suspected sediment may be evaporated on a watch-glass and tested for hæmin, when small, dark characteristic crystals are formed, shaped like little rhombic plates or rods with sharp angular ends. The hæmin test is simply made and is proof positive of blood. To the dried residue is added a few crystals of common salt, a drop of glacial acetic acid, and the mixture boiled. The crystals are seen under the microscope.

The color of the urine depends upon the amount of contained blood and the reaction of the fluid. Acid urine darkens the color, alkaline urine brightens it. If in any quantity, the urine is markedly albuminous and cloudy. When blood is derived from the bladder it usually forms clots, while blood coming from the kidney is apt to be diffused homogeneously, to be of dark-red or brown color and acid in reaction. On standing a brownish sediment is deposited. Sometimes thread-like clots are found. Blood-casts when associated, show the source of the hæmaturia to be the kidney. Acute Bright's disease, chronic interstitial nephritis, malignant growths, tuberculosis, cantharides, oil of turpentine and other powerful diuretics are causes of hæmaturia. Injuries are often followed by blood. In renal abscess and renal calculus the blood is mixed with pus. I reported a case a few years ago of death from purpura hæmorrhagica of the kidneys. Blood from the bladder is occasioned by diphtheritic and acute cystitis, calculi, carcinoma, congestion, injuries, fibroids, polypi and varicose veins about the neck of the bladder.

The urine is usually alkaline in reaction and if the hæmorrhage is occasioned by cystitis pus, mucus and triple phosphates are found. Irregular clots occur, and the color is bright-red. Blood occurring in large quantities may coagulate within the bladder.

Epithelium exists in small quantities in normal urine, and is cast off from the urinary tract as from the body surface, the intestines and the respiratory tract. It is increased in disease of the genito-urinary system, as in disease of the other surfaces named. The sources from which the epithelium is derived can not in all cases be told by the form of the cell. The large, flat, irregular squamous cells, with prominent nuclei, are derived from the bladder and the vagina and are the most distinctive cells met with in the urine. The columnar cells, elongated spindle, cylindrical, or caudate cells, with a well-

marked nucleus are derivable from any part of the urinary tract, from the pelvis of the kidneys to the urethra inclusive. Lastly, small, round or spheroidal granular epithelial cells with nucleus and nucleolus occur singly or in groups. Such cells are derived from the kidney tubules but are also found beneath the superficial layers of all the rest of urinary tract, hence in inflammations causing denudation or exfoliations are not distinctive. They occur in quantity in acute Bright's disease and often are associated with casts. Sometimes the cells adhere to the casts. In cystitis the flat, squamous cells predom-

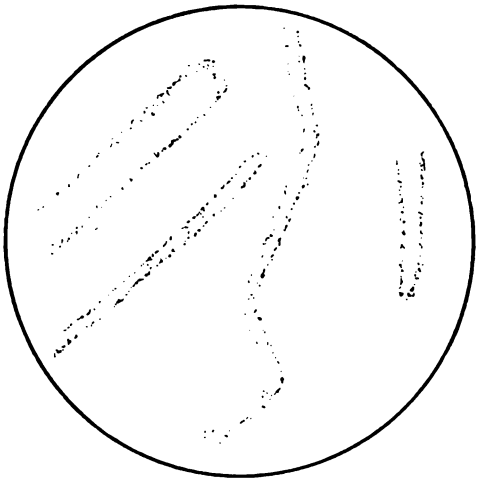

Fig. 80—Hyaline Casts.

inate over the other varieties, while in inflammations of the tubes they occur very sparsely.

CASTS.

Casts are moulds of the uriniferous tubules. The mode of their production and the material of which they are composed are not yet ascertained. From their appearance they may be separated into three classes.

(a) Clear or hyaline casts.

(b) Casts composed of histologic elements, as blood corpuscles, epithelium and pus cells.

(c) Those consisting of waste-products, the result of tissue change.

The urinary sediment having been separated by the centrifuge, a

few drops are placed upon a glass slide or in a shallow cell and well distributed. With a low power and concentrated light the field is carefully searched. Sometimes hyaline casts which are exceedingly transparent, hence liable to escape detection, are best seen in a subdued light or in shadow. They may be stained with iodine or magenta, which facilitates the search, but alters the appearance and may thus lead to wrong inferences.

Hyaline casts may be perfectly clear and homogeneous or they may show slight granular structure in parts. Sometimes a fragment

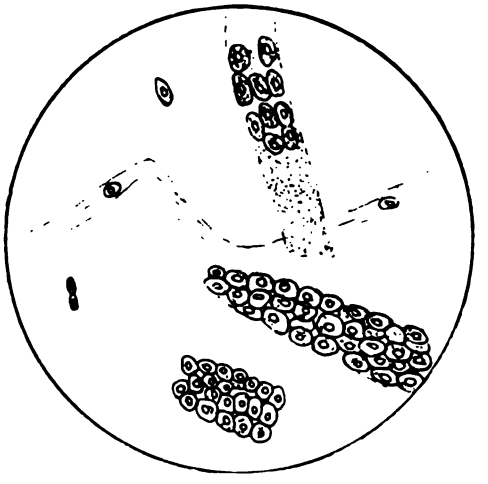

Fig. 81—Epithelial Casts.

of epithelium adheres and renders them more visible. They are very pale and transparent, of varying widths and lengths, extending in some cases clear across the field. They are only found in albuminous urine and disappear from alkaline urine on standing. Wide hyaline casts, more refracting than the above are found, which exhibit the amyloid reaction. They were formerly called waxy casts and supposed to indicate amyloid degeneration of the kidney. Sir William Roberts refutes this, and says the reaction is due to degenerative changes in the casts themselves.

Blood casts occur in cases of congestion or haemorrhage of the kidney, hence are often associated with blood in the urine. They consist of cylindrical moulds, generally short and rounded at the ends, composed of tessellated blood corpuscles.

Epithelial casts are usually hyaline moulds to which the epithelium is adherent, sparsely, in clusters, or clumps. Sometimes, however, the entire epithelial lining of a portion of the tubule seems to be exfoliated intact, giving rise to cylinders composed of intact epithelial cells. They intimate clearly a catarrhal or desquamative inflammation of the tubules and are highly important from a diagnostic standpoint.

Pus casts are rare, pus corpuscles adherent to hyaline, granular or epithelial casts are often seen.

Granular casts are frequently met with. They are moderately broad and as generally found one or both ends present a broken-off

Fig. 82—Granular Casts.

appearance. Sometimes one end is rounded, very rarely both ends. The granules vary from fine to coarse, and the colors are white, yellow and dark. The tissue elements frequently cling to their surfaces, epithelium, fat, pus corpuscles or leucocytes. They indicate chronic degenerative changes and are found in chronic parenchymatous and chronic interstitial nephritis.

Fatty casts indicate extreme chronicity and corresponding degenerative changes. They are commonly attributed to the large white kidney. The fat globules are studded over the surface of the cast in such a way as almost completely to cover it. The globules vary in size and sometimes small fatty crystals co-exist.

Bacterial casts composed of masses of micrococci indicate grave infective processes. Under the microscope they appear granular, dark in color. They do not disappear upon the addition of alkali or acid and when examined under high power their nature is apparent.

With regard to the diagnosis of the forms of kidney lesions from the variety of the cast Osler remarks: "The character of the cast is of use in the diagnosis of the form of Bright's disease, but scarcely of such extreme value as has been stated. Thus, the hyaline and granular casts are common to all varieties, the blood and epithelial casts, particularly those made up of leucocytes are most common in the acute cases."

Cylindroids, described by Thomas, occasionally occur in the urine. These are long, ribbon-like structures with branching ends, transparent, colorless and hyaline. Their exact nature and significance is undecided.

Spermatozoa are readily recognized by their shapes. The head is oblong or oval and the thin tail is directly attached. When alive they exhibit active cilia-movements. They are always found in the urine succeeding ejaculation, and are persistent in spermatorrhœa and in the urine of confirmed masturbators. They are occasionally found in acute infections and in post-epileptic states.

Bacteria. Yeast fungi and fission-fungi are found in stale urine. The latter in habitual catheter users. Ammoniacal bacteriuria is the name given to the condition resulting from ammoniacal bacterial fermentation within the bladder. It is common in catheter users. The principal agent in its production is the micrococcus ureæ.

The pus-producing organisms, as well as the bacteria of all infective diseases, may be found in the urine. Bacillus tuberculosis is found in tubercular infection of any part of the tract, and the gonococcus in urethral infection. The first is searched for in the concentrated sediment, after treating it just as sputum is treated.

Gonococci lie in pairs (diplococci) within the pus-cells. In recent infection they are abundant and easily found in the purulent secretion.

SECTION XVI.

THE FÆCES.

A complete examination of the fæces includes macroscopic, microscopic and chemic examination, and is of importance principally as a guide to the state of assimilation or mal-assimilation of the food. Such research is not always necessary for diagnostic purposes. The number and character of the movements should be inquired into and whether they are easy and natural or produced by effort and straining, whether they are followed by pain, descent of the mucous membrane, hæmorrhoids, bleeding or other unusual phenomena. In the inspection of the stool, the color, odor, quantity, consistency, form and appearance are noted.

Both the frequency of the discharges and the amount of the dejecta vary with the individual, as well as with the amount, kind and quality of the food ingested. It is influenced by age, sex, habit and occupation. While normally the bowels should move once in twenty-four hours in adults, departures in either direction cannot be pronounced unnatural. Many individuals habitually have two or three movements per day; others, a movement each forty-eight hours. Regularity and the resultant effect on the health of the individual are more to be considered than physiologic rule. It is wiser and safer not to disturb the established order and habit of the bowels, provided the health equation remains unaffected, than to attempt to establish new habits, especially in adults, but the inculcating of correctness and regularity in the child and the beneficent effects thereof, cannot be over-estimated.

In nurslings and milk-fed infants the number of stools varies from three to five in twenty-four hours, decreasing with age and mixed diet.

Constipation is a bodily condition or habit in which the amount of the fæces and the number of the movements are less than normal.

Obstipation is the temporary or permanent absence of movements due to some pathologic cause, and is generally symptomatic.

Diarrhœa is an increase in the number and amount of the dejecta, usually accompanied by a change in their character.

Painful straining at stool is called *tenesmus*, while the acute, colicky pains which accompany diarrhœa are called *tormina*.

Pain, burning and itching, succeeding stool, demand an examination of the lower bowel for the presence of fissure, ulcer, abscess or hæmorrhoids. Cancer, syphilis and tuberculosis of the lower bowel may cause great pain with evacuation. In a case of tuberculosis of the rectum, under observation, the pain is severe and lasts from one to two hours after the movement. Descent of the bowel and hæmorrhoids are readily detected by examination. Rectal prolapse in children is not infrequent.

Inspection. The normal color of the fæces is brown or yellow-brown, but varies with the food eaten. In children the milk stool is light-yellow. Chocolate and corn syrup impart a dark-brown or black color to the dejecta. Certain green vegetables, as spinach and sprouts produce green stools, due to the contained chlorophyll. The green color seen in infantile diarrhœas is due to butyric and lactic acid fermentation, acting on the bile coloring matter. In adults green stools may sometimes be attributed to a microscopic vegetable growth known as chlorococcus. Minerals such as iron, bismuth, lead and manganese, taken by the mouth, impart a black color to the stools, due to the formation of the sulphides of these metals. Mottling is not infrequent but has no special significance. Fresh blood imparts a bright-red color to the fæces, which changes to dark-brown when the matter is retained within the bowel. If retained for a considerable time the stools become black and tarry, due to hæmatin. Clay-color indicates lack of bile. The normal color depends upon the amount of bile-pigment present and the amount of change the latter has undergone, the results being bilirubin, biliverdin and their oxydation product choletelin, or their reduction product hydrobilirubin and stercobilin.

The *fœtor* depends in a large measure on the presence of indol and skatol which are the outcome of putrid bacterial activity: the decomposition of albuminoid substances, and upon the presence of certain fatty acids. Sulphuretted hydrogen, traces of hydrogen phosphide, ammonia and organic bases contribute to the odor. In some catarrhal diseases and in dysentery the odor is excessively offensive.

In gangrene it may be putrefactive or cadaverous. Infantile and summer diarrhœas have a sour odor due to fermentative changes.

The *quantity* of fæces evacuated in a day depends upon the diet, being greater with a vegetable or starchy diet than on meat diet. The average quantity is about 150 grams a day, varying between 60 and 250 grams (Yeo), of which about 75 per cent. is liquid. The more rapid the passage of the ingesta through the bowels, the greater the amount of fluid which remains with the fæces, which explains the diminished consistency of stools caused by intestinal irritants and substances which stimulate peristalsis. In diarrhœal diseases the amount of fluid is much increased.

The *consistency* of the normal stool is such that the passages are formed and firm. A thick, mushy consistency is more usual in summer time when vegetables and fruits are largely indulged in. As said, the consistency increases with retention. Experiments made by myself to determine the time required for non-irritating substances to pass through the normally active healthy bowel, seemed to establish thirty-six hours as a fair average. H. C. Wood states, on what authority I know not, that only four hours are required for the contents of the bowel to pass from the pylorus to the cæcum.

Although the reaction of the large intestine is markedly acid, due to acid fermentation, yet the *reaction* of normal fæces is neutral, sometimes alkaline, more rarely acid. The alkalinity is due to ammoniacal fermentation, and is increased by meat diet, the acidity to lactic and butyric acid fermentation, but sometimes acetic and propionic acids are found. Vegetable diet assists in their production. Much mucus renders the stools alkaline. The acid reaction of nurslings and milk-fed infants is due to lactic and free fatty acids.

Composition. The fæces are composed of the undigested parts of the food, the useless and injurious portions of the various secretions, decomposition and microbic products and gases.

Among the first are found yellow elastic tissue, tendons, nuclein, epidermic and horny substances, vegetable fiber, chlorophyll, gums, resins, cellulose from vegetable sources, particles of food which, from imperfect mastication have not been acted upon by the digestive juices, alimentary substances which are assimilable, but of which an excess has been taken, especially fats and starches, insoluble salts, as silicates, sulphates and phosphates of calcium and ammonium-magnesium phosphate.

In mixed diet the waste secretions exceed the residue derived from the ingesta, and consist of intestinal mucus, epithelial cells, substances which failed of absorption, as decomposed bile, coloring matters and inorganic salts; free fatty acids from acetic up to palmitic acid, butyric, lactic, cholalic acid and cholesterin; indol, skatol, cresol, phenol, leucin, tyrosin, each may appear under certain conditions. The gases expelled are CO_2, CH_4, H, N, H_2S. The N is derived from swallowed air, the rest arise in the course of decomposition.

The *meconium* is the name given to the contents of the intestine which accumulate during fœtal life. It is usually evacuated soon after birth. Its color is dark-green or brown and its consistency salve-like or tarry. It is acid in reaction and devoid of fæcal odor. It consists of epithelial cells in abundance, cholesterin, fats, fatty acids, mucin and bile-coloring matters.

The stools of milk-fed infants contain fats, fatty acids, casein, epithelial cells, lactate of calcium, mucin, coloring matters and bacteria. In infants fed on cow's milk, the casein is much increased over that of breast-fed infants.

Acholic stools are wanting in bile. The color is blue-clay or chalk-paste. They are very fœtid and contain large quantities of fat. Suppression of the pancreatic secretion is followed by fatty stools, and their persistence should be viewed with grave suspicion.

Diarrhœal stools contain excess of water. When due to catarrhal conditions they contain epithelium and much mucus. In acute cases the latter imparts character to the passages, which are named mucous stools. Medicines which hasten peristalsis produce the same watery evacuations. Further, the activity of the peristalsis prevents absorption, hence hurries down much unassimilated food substance.

The characteristic typhoid stool is offensive, abundant, thin, grayish-yellow, granular, and in appearance and consistency resembles pea-soup. The reaction is alkaline. They sometimes contain blood, shreds of necrotic tissue, and, generally, ammonium-magnesium phosphate. On standing they separate into two layers, an upper, serous layer containing albumin, and a lower layer containing the residue, food and epithelium. After the end of the first week is found the Eberth bacillus and a poisonous base known as typhotoxin. Blood and necrotic shreds are recognized by the microscope.

Cholera stools are ever likened to rice water: thin, turbid or translucent. Koch's spirillum or coma bacillus abounds, especially

early in the disease. Epithelial cells in abundance are seen under the microscope. The discharges contain *nitrites* which, with the indol of the stool, gives a blood-red color on the addition of dilute sulphuric acid. The reaction is diagnostic.

The discharges of dysentery are peculiarly fœtid, voided with great tenesmus, tinged with blood, contain much mucus, and, in old cases, fragments or sloughs of mucous membrane and pus. Amœba coli characterize the form known as tropical dysentery. Fresh dejecta must be examined upon a warm stage in order to demonstrate their presence. The fibrinous casts and shreds of mucus should be examined by floating in water.

Gall-stones and enteroliths are sometimes found. When gall-stones are suspected, the stools should be passed into a cheese-cloth "crab-net" over a wire frame, placed in the closet, then washed in running water. The stones are recognized by their hardness, shiny appearance, color and facets, giving them an uncut crystal appearance.

Enteroliths are solidified food concretions of varying, usually small, size. Seeds, animal parasites, eggs and larvæ are often found in the fæces.

Pus occurs in cases of abscess rupturing into the tract or in destructive inflammations of the bowel itself, as in dysentery.

In cases of tuberculous, syphilitic or cancerous ulceration pus is mixed with tissue shreds and blood.

Microscopic Examination. When very thin the fæces can be examined without dilution, or a small portion may be thinned with water, spread on a cover glass, dried, fixed and stained in the usual way. Bacteria, pus, blood, epithelial cells, crystals, and detritus are thus recognized. In conditions of intestinal suppuration, leucocytes are found. The crystalline substances encountered are fat crystals, phosphatic crystals, oxalate of lime, sulphate of calcium and occasionally others.

SECTION XVII.

THE ROENTGEN RAY IN DIAGNOSIS.

The X-ray contributes very considerably to the science of diagnosis and in some cases, at least, affords aid at just the point where other methods are imperfect or totally inadequate. Hence it is to be regretted that the system is not of more universal applicability. In private practice its use is not yet extensive, but at present all large hospitals and most smaller institutions, as well as many physicians' offices, are equipped with the necessary apparatus.

Since, however, for a long time to come the greater number of examinations for ascertaining the physical condition of patients must be conducted without its aid, practitioners will do well to cultivate and practise the older methods of diagnosis, even in the instances to be described in which its use affords us most comfort by reason of its certainty.

The static machines constructed for office use are equipped with the necessary X-ray apparatus. They are expensive, bulky, stationary, but efficient. At present the work is generally confined to men of special training, but experience is the only requisite.

For purposes of diagnosis in diseases of the chest a high penetrating power is unnecessary and less efficient than one of lower power since the latter renders less transparent the organs which we most wish to examine, by reason of such solid organs obstructing the softer, less penetrating rays. This is brought about by using Crooke's tubes of less perfect vacuum than those necessary for viewing the bones and large joints. Such tubes are technically called soft tubes.

The Examination. The examination may be direct, by the use of the fluoroscope, to which proceeding the name *radioscopy*, or Röntgoscopy, is given; or the examination may be indirect, by making radiographs or plates from which prints are made. The latter process is called *radiography*.

A fluoroscope is simply a piece of card-board coated with a fluor-

escent salt—platinocyanide of barium is the best—and fitted into a frame like the stereoscope.

In general hospital work the examinations are made almost entirely with the fluoroscope as the results are immediate, and in the hand of a practised examiner much more information upon certain necessary points is vouchsafed than by the pictures. But the direct

Fig. 83—Normal Chest. The natural bulge in aorta referred to in text is seen at B.

examination requires that the patient be submitted to the ordeal for a much longer time than is necessary to secure the photographic picture, and is tiresome likewise to the examiner. In cases of severe illness this is a point worthy of consideration. On the other hand it is the only way in which pulsations, the movements of organs, and changes in the position of fluids may be appreciated.

As intimated, experience and good judgment play no small part in forming correct conclusions by this method, since only by practice and experience the relative position, size and qualities of the organs

in the shadow pictures can be learned, as all these vary with the distance between patient and light, and other factors.

Radiography is most useful in doubtful and disputed cases; the prints have a certain medico-legal value, can be used for measurements and comparisons, and when successive pictures are obtained show positive results of treatment or of the advance of disease, and furnish us with permanent records.

Since the improvements in apparatus and technique, the necessary exposure has been so shortened that at present satisfactory pictures are obtained by exposures of less than a second (von Ziemssen).

Fluoroscopic Examination; Radioscopy. The examination is conducted in a dark room and is simplest and most satisfactory with the patient erect, as in this position only are the normal relations preserved. The Crooke's tube is placed behind the patient in the median line at a distance of about two feet, on a level with the part to be examined. A fenestrated, adjustable metallic screen, or a Beck's diaphragm, placed between the tube and the patient is useful for concentrating the light on a particular point and for cutting off extraneous rays. In case the patient is unable to stand, the tube is adjusted under a canvas cot on which he rests. The fluoroscope is placed directly in front of the chest walls.

Röntgen ray burns are the result of too long or too oft-repeated exposures. Restricting the single exposures to five minutes, and not repeating these oftener than at intervals of every three days, is said to be a safe limit.

THE THORAX.

The healthy lungs do not perceptibly intercept the rays hence their image appears only as a faint shadow. The vertebral column, the ribs, clavicles and sternum are plainly seen. The pericardium, the outlines of the heart and its movements are distinct. The great vessels are less distinct, but visible. The movements of the diaphragm are distinct and when restrained the limitation may be detected by the practised observer. In children, and generally in adults, the bifurcation of the trachea may be seen.

The apex of the heart shows sharply during deep inspiration. A bend or protuberance upon the left of the aorta, above the pericardial sac, resembling an aneurysm is often seen and might be mistaken readily for such a growth.

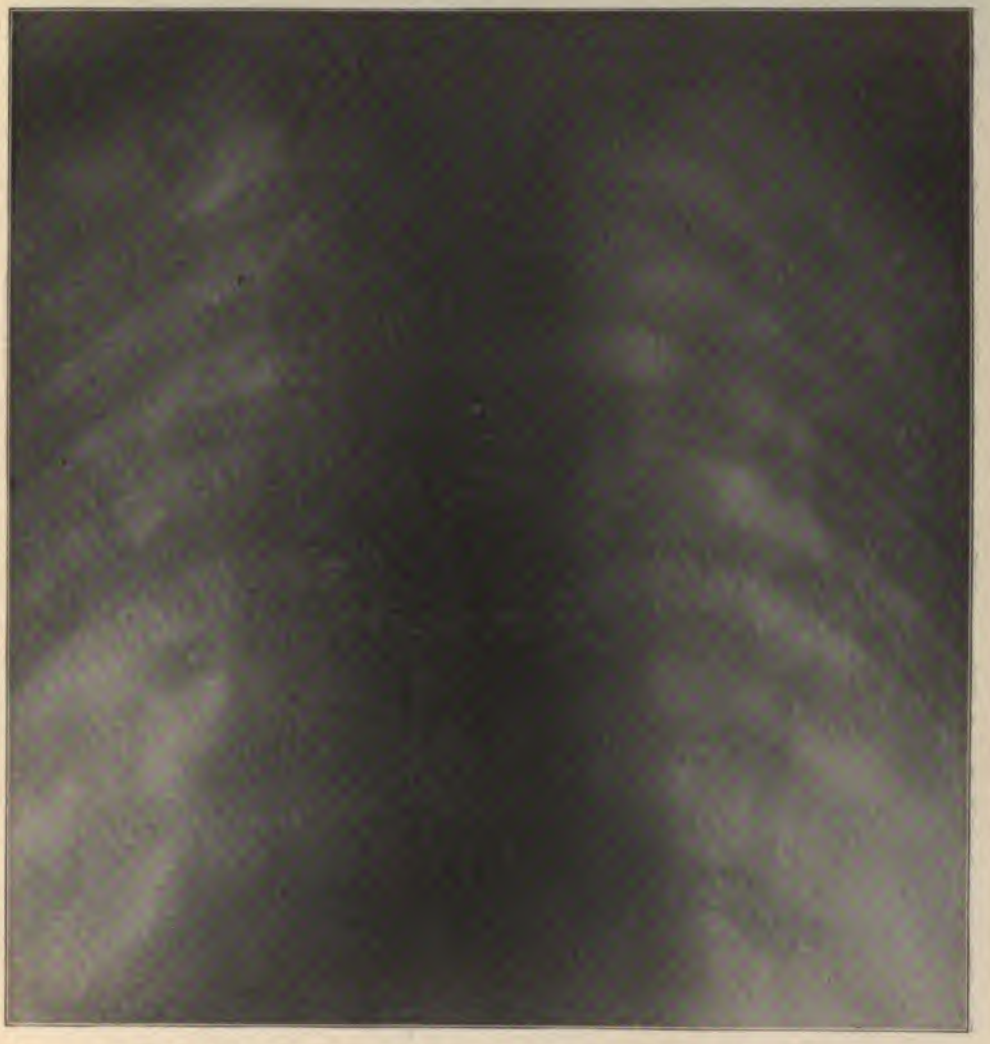

Fig. 84—Small Aenurysm of Aorta. Johns Hopkins Hosp., Dr. Baetjer.

Fig. 85—Disseminated Pulmonary Tuberculosis, both lungs. Johns Hopkins Hospital, F. H. Baetjer.

The unequal heights of the two sides of the diaphragm is strikingly seen, the right side being always the higher. This shows best during deep inspiration. In case of hypertrophy of the heart the left diaphragm is sunken below the normal plane, while in early tuberculosis Williams finds the structure abnormally high.

Diseases, injuries or malformations of the vertebræ, supernumerary ribs, or foreign bodies within the thorax are easily recognized.

Cardiac hypertrophies and dilatations not only can be recognized but differentiated (von Ziemssen). Displacements, effusions into the pericardium and aneurysms of the organ are made out, as are also mediastinal tumors.

While the healthy lungs give almost no image, yet diseased conditions are fairly well brought out by the rays. Pulmonic solidifications, especially deep-seated foci of pneumonia almost impossible to demonstrate otherwise, appear, as do tuberculous solidifications, vomicæ, abscesses, bronchiectasis, atelectasis and calcifications.

Cavities with thickened walls are seen as shadows surrounded by lighter areas. Incipient tubercular lesions show but little, and it is doubtful if they can be recognized by the rays before they are demonstrable by other means.

Effusions into the pleural cavities are markedly opaque, and the movements to which they are subject are strikingly shown. Serous effusions appear more opaque than do those of pus. The boundary line between effusion and air in hydropneumothorax is distinct. Pulmonary retraction resulting from old pleurisy, as well as thickening and deposits upon the pleural membrane, all show in the plates.

Irregular movements of the diaphragm and restrictions of its excursions are easily studied. The diaphragm separates from the heart during deep inspiration.

During forced inspiration the lung shadow is diminished. In emphysema the lung shadow is fainter still than in normal lungs. The heart obstructs the shadow of that portion of the lungs which it covers.

Sclerosis of the deep vessels can be recognized only by the x-ray picture. The position, size, shape and type of intrathoracic aneurysms are better told by the skiagraph than by percussion and auscultation, and repeated instances are recorded in which they have been discovered by this means when their presence was unsuspected. Pulsation distinguishes them from solid tumors of the same area.

THE ABDOMEN.

In diseases of the abdomen a somewhat harder tube than that used in chest work is desirable. Skiagraphy gives the best results. The solid liver shows best; the intestines are discernible only occasionally, but their contents show. The triangular psoas muscle can be seen. The outline of the stomach is shown by coating its walls with a substance impenetrable by the rays, as subnitrate of bismuth, or by the introduction of a flexible steel wire covered with rubber.

Tumors of the stomach show only when of considerable density.

Radiographs of the intestines are obtained in the same manner as has been suggested for taking skiagraphs of the stomach. One ounce of bismuth subnitrate is suspended in a quart of milk. The skiagraph of the stomach may be taken immediately after its ingestion. Six hours later the picture of the small intestine is taken; twelve to twenty-four hours later the colon shows best in the print.

Recent examiners are all in accord in stating that the position of the stomach as described in the text-books is incorrect. The vertical position, as referred to in the description of the organ in the section on anatomy, and the position which has usually been considered and described as ptosis or moderate prolapse, are most often observed. The same may be said of the intestines; what formerly has been regarded as prolapse of the colon is more commonly found than the so-called normal position.

The examinations show that dilatation and prolapse are frequently met with, especially in women.

More extended research will probably change many of our prevalent ideas on these topics.

The spleen appears indistinctly, as do also the kidneys. They appear in the radiographs but are seldom seen by the fluoroscope.

Diseases, fractures and displacements of the lumbar vertebræ; calculi of kidneys, ureter, bladder and prostate, and those of the hepatic duct and gall bladder are demonstrable.

The size, shape and position of the liver are easily studied by the rays, and abscess, when present, is revealed.

Biliary calculi are difficult to show in the picture and much depends on their composition, but in recent years the difficulties have been successfully overcome.

Hydronephrosis and cysts of the kidney have been demonstrated,

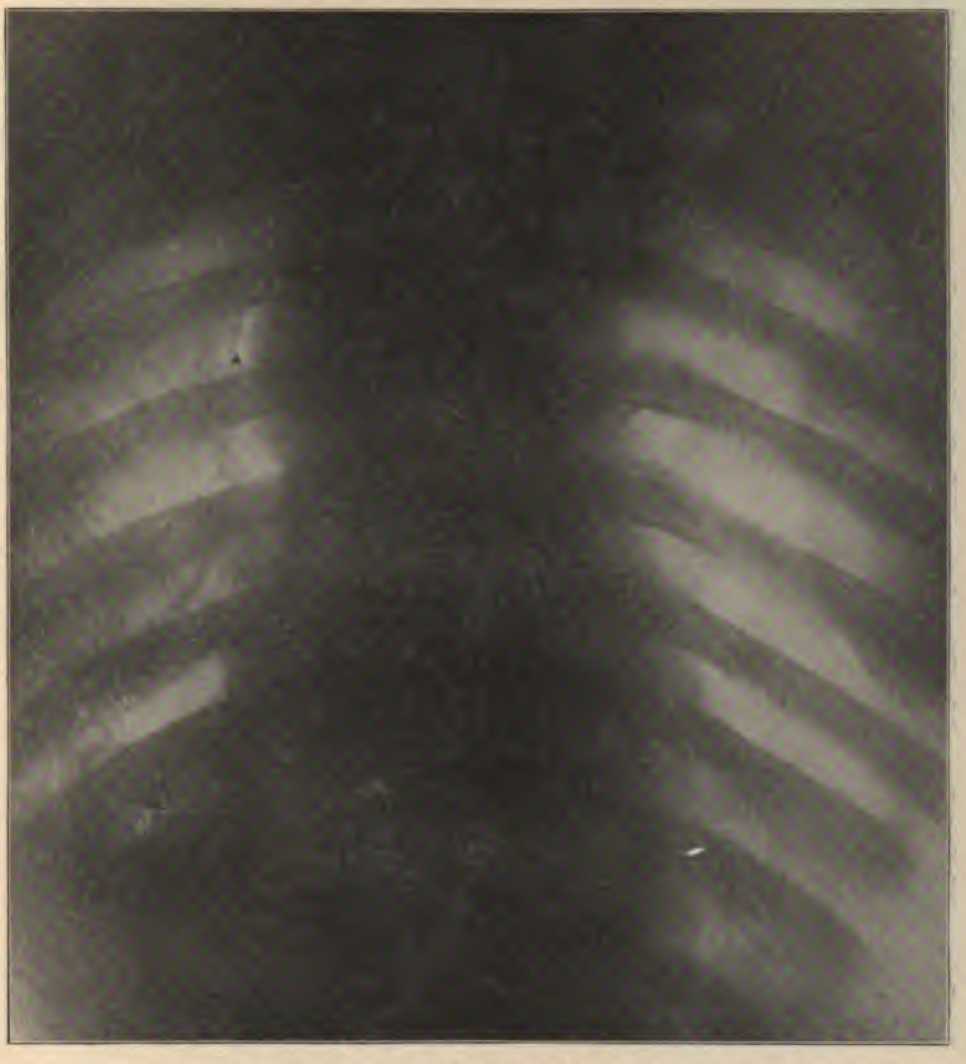

Fig. 86—Aneurysm. Johns Hopkins Hospital, Dr. Baetjer.

Fig. 87—Aneurysm of Aorta. Johns Hopkins Hospital, F. H. Baetjer.

but it is in calculi of these organs that the pictures are of paramount usefulness, since the absence from a good plate of indications of their existence justifies a negative diagnosis in most cases. Ureteral and vesical calculi also can be skiagraphed.

The Röntgen ray diagnosis of renal calculi is more satisfactory in children than in adults. The most important factors are the size and composition of the stone, the size of the subject under examination, and the condition of the kidney, according to Smart. He says that pure uric acid stones are the most difficult to detect; pure phosphatic stones ranking next. A combination of uric acid with phosphate or oxalate of calcium is more easily detected, while pure oxalate stones are the most opaque to the rays. Occasionally a collection of pus or induration of the organ may obscure the shadow. The intestines of the subject should be well emptied by enemas and purgatives, and all clothing removed from the parts of the body which are to appear in the negative.

Breathing should be restricted by an abdominal binder in the case of children, as the movements of the kidneys interfere with the sharpness of the pictures. Adults may be placed face downwards on a canvas cot, the weight of the body being sufficient to limit the respiratory excursions. In this case the tube is placed under the cot, and the plate on the patient's back, as before described. Both kidneys and both ureters should be included in the picture. In case of doubt, the exposure should be repeated a few days later, the conditions being as nearly similar as possible.

Foreign bodies in any part of the abdomen are easily located, especially if metallic, as nails, tacks, coins and the Murphy button.

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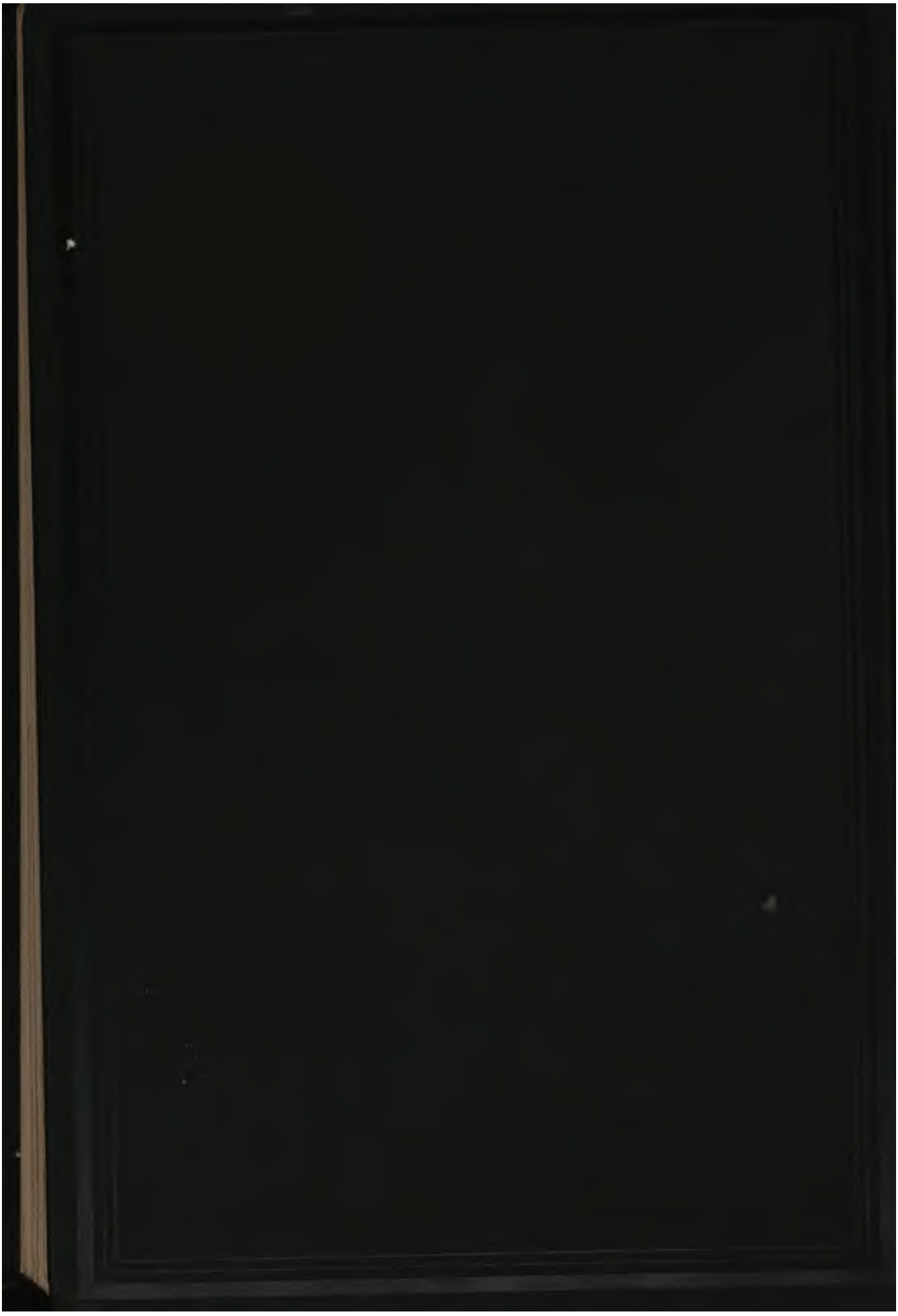
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